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THE FIRST TEN MINUTES

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I SOMETIMES WONDER if obstetrics shouldn't have joined up with pædiatrics instead of gynæcology, and if—in whoring after the glamour of surgery—it hasn't missed the crux of the matter. For the crux surely is not just to deliver a woman but to get a new citizen sound in mind and body. True enough, as the current maternal mortality rate shows, our obstetrical obsession with the mother has been eminently successful. But the fate of the baby is another kettle of fish. Deaths in the first week of life still rank third in Nova Scotia after cardiovascular disease and cancer. We obstetricians must accept responsibility for all but a minor fraction of these since, in effect, most neonatal deaths are obstetrical deaths, conditioned by what happens during pregnancy, labour and the immediate neonatal period, when the situation is still in our hands.

Many of these deaths are due to prematurity. Some are due to fetal abnormalities. Some to complications of and difficulties with labour that even the best skills cannot completely circumvent. But how many of these neonatal deaths, how much neonatal disability of a more or less serious nature, not only in the premature but in the mature, result from a neglect of the baby during that vital first 10 minutes of its life, when it is trying—often far more desperately than we realize—to accommodate itself to extrauterine existence?

To what extent is our obstetrical philosophy responsible for this neglect? What seems to happen so often is this: immediately the baby is born its cord is clamped and cut, it is handed to a nurse who puts it in a cot at the far corner of the delivery room, or in one actually outside the delivery room, while the obstetrician proceeds to sew up the episiotomy. Presently someone comes in to say that the baby is either not breathing well or has stopped breathing. After that a nerve-wracking 10 or 15 minutes of resuscitation ensues, until the grey-blue baby responds. Sometimes it doesn't. Even if it does, we cannot help remembering that the pædiatric pathologists say that an apnœa that lasts so long does permanent damage to highly developed infantile cells—particularly in the brain. To this end it is suggested that we give some thought to the following factors:

A. *Not to let the baby out of our hands until it is breathing properly and has a good pink colour all over its body.*—It is not enough to wait until it cries. I have known babies to die of asphyxia despite what seemed a satisfactory birth-cry. The baby should be breathing quietly and regularly and the pink colour should have extended to its hands and feet. In the meantime, it should have cried *well.* The first requisite for proper breathing is a clear airway. To achieve this in any baby I know of nothing better than to hold it upside down and allow gravity to drain it, or aid in any other efforts to drain it.

This doesn't mean holding the baby up by its feet. A baby's skin is greasy and slippery, and so are rubber gloves. One has a far safer contol if it is grasped over both shoulders with the fingers of the left hand, its back being against the forearm. In this position it can be securely held without danger of slipping, and the right hand is left free for other purposes (see Fig. 4). I make a practice of holding all babies upside down in this fashion until they are completely drained of amniotic fluid. This process takes five minutes *by the clock.* It is surprising how frequently some amniotic fluid drains away as long as four minutes after birth, even from the lustiest baby.

I have heard it argued that the upside down position, being an unnatural one, may cause the baby harm through encouraging intracranial hæmorrhage, etc. Those making this statement seem to forget that almost all babies have been standing on their heads *in utero* for months and are thus fully accommodated to the position.

Furthermore, some pædiatricians claim that all newborn babies are better off spending the first few hours of their life on the slant with their heads down.

While the baby is being held in this position, gravity should be aided wherever necessary by flicking any thick mucus from its pharynx with the gloved finger, and by milking the trachea on the outside. If this is done, pharyngeal suction with a sucker is practically never needed. If the above-described method of drainage fails to bring a response, it is better to proceed at once to catheterization of the trachea with a laryngoscope. Occasionally, if the baby's mouth is big enough, the catheter can be guided by a forefinger into the larynx. So often the baby is small or premature, in which case catheterization without the laryngoscope becomes very much a matter of guesswork.

My experience with resuscitating machines has not been happy. Over the years we have employed several, always to end up with frustration and disillusionment. Not only have they a habit of being out of order when most urgently needed, but some of them do damage. In large hospitals where they are being constantly used, they are more apt to be in working order and—if of a simple type like the Mann—probably serve a useful function. In hospitals where they might be required only once a fortnight or so, lack of familiarity with their mechanism is likely to prove them more a menace than a help. Catheterizing the baby's larynx through a laryngoscope, attaching an oxygen bag to the catheter, and creating negative and positive pressure with the hand against the bag has the virtue of simplicity and availability.

B. *To leave the baby attached to its mother until it has received all its blood from the placenta.*—As stated previously, it seems to be common practice to put two clamps on the cord immediately the baby is born, cut between them, and hand the baby over to a nurse. At one of the less admirable stages of my career, I used to abstract blood from placentæ for transfusions elsewhere. It proved a useful source at a time when blood was very scarce in Halifax. On such occasions I was able to extract anything from 50-250 c.c. of blood per placenta. Since to a baby of seven and a half pounds 50 c.c. represents a transfusion of 1,000 c.c. to an adult of 150 pounds, this caused a considerable loss to the infant. So later I went back to my earlier practice of not separating the baby from its mother until the cord had stopped pulsating and changed colour from a pearly grey to a dirty white.

Some of the arguments brought forward against this practice, by those who immediately sever the baby, constitute a rather interesting commentary on the manner in which the human mind works when rationalizing its own actions. Before we knew that fatal jaundice in the newborn was due to Rh factors, I was assured that it was allowing the placental blood to continue to drain into the baby that caused it. The two arguments I now hear are (1) that it may hamper the baby's heart by putting too big a load on its circulation and (2) it may increase the tendency to intracranial hæmorrhage. These strangely illogical ratiocinations utterly disregard the fact that, until the human race began to chip flints, there was no way of severing the umbilical cord except with the teeth. It seems likely therefore that for over 99.9% of our existence as a species, the baby stayed attached to the placenta until all the blood had drained out of it. If this had been an inimical factor, surely the race would have died out æons ago.

It would seem therefore good practice for us to follow nature in this matter and let the baby have its immemorial meed of blood. Especially should this be the case in premature babies, whose digestive capacities are not very efficient in the first week of life and who can do with this extra and so easily assimilable protein. The only exception to this rule would be the baby of the Rh negative mother, whose blood contains antibodies; its cord should be tied as soon as it is born.

In late years there has grown up a practice—probably the result of hospital delivery—of clamping the cord twice, cutting between the clamps, and then handing the baby to a nurse who later on ties it. Is this sound practice? Everybody who has used clamps surgically with any frequency has seen them slip. I saw this happen to a baby who lost quite a lot of blood from its cord before the accident was discovered. Furthermore, if any type of resuscitation has to be done, the clamp can get very much in the way and become a decided impediment to efficiency. It seems to me that we owe it to this newborn child to take the few extra seconds required to tie the cord with tape, cut it, dab it

with a bit of gauze, and then watch it for a minute to see that it is dry.

C. *The newborn baby should be so placed in the delivery room as to be in full view of obstetrician and nurses.*—Most delivery tables do not provide a place on which to lay the newborn baby, with the result that it must either be laid on the mother's abdomen, a rather insecure locus, or held by the obstetrician. In the latter case, if the woman is being delivered in the lithotomy position, the nearest prop is the floor—and greasy babies can slip through rubber gloves. Even in those tables that do provide a platform that can be lifted into position under the buttocks, this platform is small and insecure, and the baby has to be watched lest it roll off. Where such a platform is available it should be used and the baby—after it is thoroughly drained—laid on it to await the cessation of cord pulsation. It is my own practice in the meantime to sew up the episiotomy. The assistant holds the cord and membranes up out of the wound which can then be made quite clearly visible throughout its entire extent. The cord does not become a problem unless it is unusually short.

The great advantage of using the type of platform thus described is that the baby remains in direct view and immediately under the obstetrician's eyes for from 10 to 15 minutes—the *important* 10 or 15 minutes. Anything untoward in its appearance or behaviour is immediately evident. Some babies, especially those whose mothers have had a lot of sedative and anæsthetic, will breathe and cry on being born, and then stop doing either. Such a baby rapidly becomes a sinister bluey-grey, constituting in a matter of minutes a very serious resuscitation problem. Such babies have died. They stand a better chance if they are positioned so that the obstetrician cannot help but be the first to note the evil sign. It may take a sluggish and doped baby up to 15 minutes to make the full response to life, requiring constant stimulation and attention in the meantime.

Perhaps we don't realize, as we should, that this poorly-responding baby may be undergoing serious and permanent damage to its brain because of hypoxia. Not only does this seem to be a fact, but a considerable number of these babies either die in the first few days of life or become serious nursing problems. It is therefore of the utmost importance that this type of baby be thoroughly oxygenated during its first 10 minutes. The best way to oxygenate it is to keep it breathing, and it breathes deeper if it is crying. All this can best be encompassed by the obstetrician himself and not by some less skilled person. He can only do this effectively if he has the baby directly in front of him.

We have developed a light carriage (see Figs. 1 and 2) which can replace the table platform;

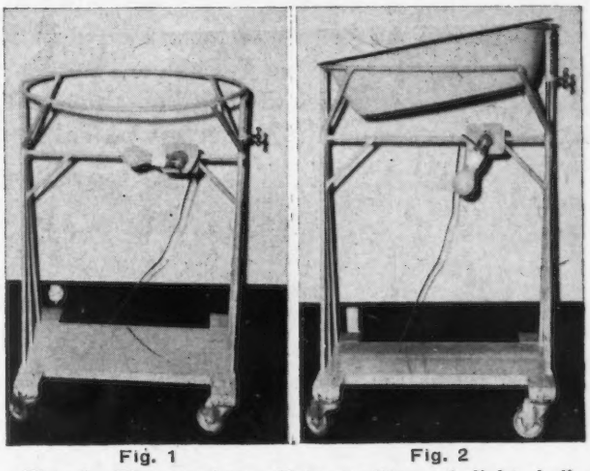

Fig. 1 Fig. 2

Fig. 1.—The carriage. Note position of light bulb.
Fig. 2.—The top tilted. Note screw handle at side to hold arc of rim in position, and range of change of position of light bulb.

it has other advantages, especially that of safety, to the baby. A large basin fits into the ring at the top, within which the baby is absolutely secure and out of which it cannot roll. One end of the ring lifts up, allowing the basin to be tilted so that the baby can lie with its head lower than the rest of its body. The carriage runs on large, ball-bearing casters, so that it can be pushed easily and noiselessly about the delivery room. Underneath the basin is an electric light on a swivel which can be moved closer to or farther away from the bottom of the basin and so heat the latter.

Just before delivery of the head the heated carriage—draped with a sterile sheet that is padded in the part that lies over the bottom of the basin, thus diffusing the heat—is pushed up against the delivery table under the mother's buttocks. The seated obstetrician is able to push his knees in under the basin and so sit close to the perineum (Fig. 3). The first carriage we made had wooden sides which had the effect of backing the obstetrician too far off from the scene of action.

When the baby is born it is held upside down to drain over the basin, so that if it slips it has not far to fall (see Fig. 4). When it is thoroughly drained it is laid in the basin and kept there

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under immediate scrutiny until the episiotomy is sewn up, and its cord has stopped pulsating and been tied and severed. The carriage is then pushed around to the side of the delivery table so that the mother can see the baby. Because of the presence of the carriage, it may be necessary to stand up to put in the top one or two sutures in the vaginal wall, but the remainder can be inserted sitting down. One soon gets used to it.

Its other advantages are: (1) it can be quickly moved to an adjacent room if a serious resuscitation problem is present. (2) The baby does not have to be picked up and carried to the nursery, but can be pushed there in the carriage—a considerable advantage in previously anoxic, or damaged babies, who should be handled as little as possible in the first few hours of life. It is of so simple construction that any good metal-worker can make it. Never since adopting delivery in lithotomy have I felt such a sense of security with regard to the baby as since using this carriage. It gives the baby absolute safety of position, keeps it in full and immediate view of the obstetrician, obviates the necessity of cutting the cord before it stops pulsating, and avoids unnecessary handling—all of which are benefits which amply compensate for any slight awkwardness entailed in its intrusion between obstetrician and perineum.

D. *The problem baby.*—This type of baby resolves itself into five common groups: (1) the premature, (2) the damaged, (3) the doped, (4) the forceps, and (5) the Cæsarean. While the needs of these groups vary, there are three desiderata more or less common to all: (a) they should be thoroughly drained, (b) they should be made to breathe as soon as possible, and (c) they should be kept warm. Even in the most torrid delivery room a baby will lose heat rapidly and dangerously if it is not breathing. This can be obviated to some extent by wrapping it in a thick towel to avoid evaporation from its skin, or by returning it to the sort of medium from whence it came—a warm bath. If it has to be intubated, the warm bath is out; but it is better to wrap it in a warm dry towel during this procedure than leave it naked. I emphasize the word "dry" deliberately. To anyone who thinks a wet warm towel is better I advise the following personal experiment: Take a warm bath and when finished wrap yourself first in a wet, warm towel—stay in it for two minutes—and then try a warm dry one. The baby whose temperature has dropped seriously while being resuscitated may make an immediate recovery, but it is apt to become seriously ill or die within a week or so.

The premature.—The three desiderata just mentioned apply particularly to this type of

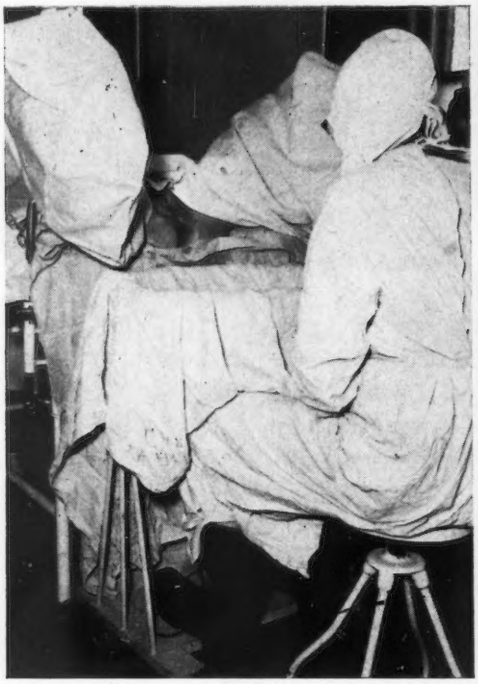

Fig. 3

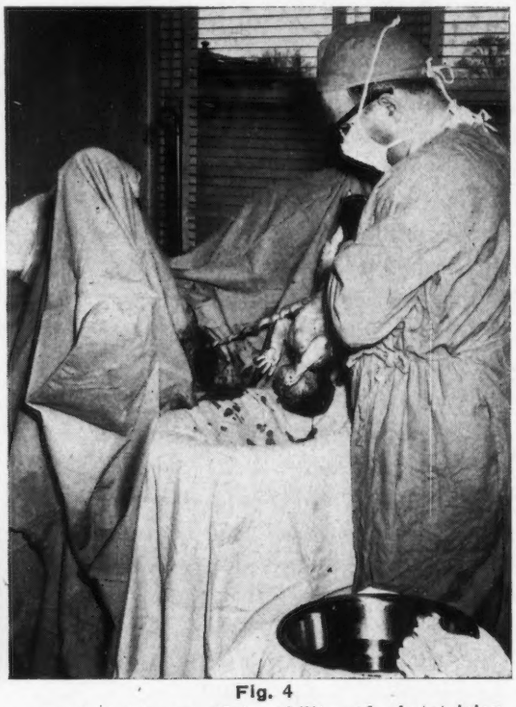

Fig. 4

Fig. 3.—The carriage in position under woman's buttocks. Note ability of obstetrician to put knees and feet under basin and so sit closer to perineum. Fig. 4.—Baby being held upside down over basin while pharynx is being cleared with finger.

baby. I believe it is better to drain it by holding it in the upside-down position previously described, than to lay it on its side—even with its head down—and have to keep constantly sucking amniotic fluid and mucus from its pharynx. After using the catheter several times this secretion takes on a sinister pinkish colour which means that the delicate mucous membrane has been breached, thus providing an entry for bacteria which can from such a lodgement push their way more readily down the respiratory tract. If these babies are drained upside-down for five minutes they will seldom need suction: gravity works continuously and gently.

It is a good practice to wrap a dry towel around the premature the moment it is born to avoid loss of heat from surface evaporation. As soon as it is drained and breathing properly it should be placed in the heated carriage, and remain there until the cord has stopped pulsating and all the blood from the placenta gone into it. Let me repeat: *Of all babies the premature should not be detached from its mother until it has got all the blood nature intended for it.* Pædiatricians like to keep the premature's stomach empty for a day or so and this extra blood is a hostage against hunger. Finally, the premature should be handled with the greatest gentleness; the more premature it is, the more need to be gentle. It is a frail and fragile thing, its life tenuous and uncertain—but it has its own powers of survival, powers we may actually hamper if we try to aid them too vigorously. When the cord has been tied, the baby should be placed immediately in an incubator, which should have been brought to the delivery room previously.

The damaged.—What was said about gentleness in handling the premature is even more pertinent here. This baby is suffering from shock, the result of a long and arduous labour, or instrumental or other complicated delivery. The mother probably had a fair amount of sedative and—if the delivery was instrumental or complicated—something approaching a surgical general anæsthesia. Therefore, in addition to being damaged it is doped. This catches one on the horns of a dilemma: the doped baby needs to be stimulated, but the shocked baby is made worse by stimulation. Furthermore, in a fairly large percentage of these cases there have been attempts at inspiration before birth, with the result that thick vaginal secretion has got into the larynx. It is therefore a matter of urgency to clear the airway to prevent a further descent of this bacteria-laden material with the first respiratory efforts.

My own preference in such cases is the previously described upside down drainage, flicking the gloved finger in and out of the pharynx and milking the trachea. While it is arguable that this damaged baby should not be held in the upside-down position for fear of increasing any cerebral hæmorrhage, I feel that this is a risk worth taking if the airways can be quickly cleared without having to resort to intubation. If this does not work and it is obvious that respiratory efforts are being hampered by airway blockage, it is better to intubate too early than too late—preferably using the laryngoscope.

Once the airway is cleared most of these damaged babies will breathe. Gentle pressure on the chest will help. Or if a catheter has been put into the trachea it can be attached to a bag of oxygen on which positive and negative pressure can be made with the hand, or it can be attached to a resuscitating machine. This baby should be given oxygen as soon as it starts to breathe, and this should be continued until it is a good colour all over and has well recovered. It should be moved and handled as little as possible for the next day or two. As long as it is not breathing well it should be prevented from losing body heat.

The doped.—Where the mother has had much sedation, finishing with a general anæsthetic of almost surgical depth, her baby is likely to be sluggish in its response to life. So is it likely to be in those cases where the mother is pushed quickly under a general anæsthetic to keep the baby back until the doctor arrives. Indeed, I have known of babies who never did breathe because of this reprehensible delaying practice, for which there is no justification either in ethics or common sense.

The doped baby *per se* is a different problem from the two previously discussed. It requires constant stimulation to keep it breathing, and should be given oxygen from the beginning. Some hold that no newborn baby should be slapped or tickled. This is unquestionably true of the premature and the damaged, but is not this doped baby the same problem as the adult who has taken an overdose of opiate? In the handling of the latter is it not our practice to keep them on their feet and walking and, to

slap their faces when they tend to fall asleep? I do not suggest that the doped baby be pummelled and beaten, but that it should be kept breathing by painful stimulation until it has taken a thorough grip on life would seem the rational approach to the problem.

We have been experimenting with normorphine (Nalline) in the type of case where the mother was sedated with such opium-like derivatives as morphine, heroin, or demerol. An attempt is made to anticipate trouble by giving the doped mother a 5 mgm. ampoule shortly before the baby is born. If that has not been done, one-tenth that amount is injected into the baby's umbilical vein. This drug has no effect where the doping is due to barbiturates or general anæsthesia. So far our results have been mixed and inconclusive: it has seemed to work in some and has had little effect in others. We are continuing the experiment. We have given up the use of alpha lobeline.

Fewer babies of this type would become resuscitation problems if we were more perspicacious in our choice and employment of sedation and anæsthesia; also if we had the baby's welfare as much in mind as the mother's comfort. If the woman is given such drugs as heroin, demerol or nisentyl to procure rest during a protracted first stage, or just at the end of the first stage when the pain of cervical dilatation is at its worst, the effects have usually passed off by the time the baby is born. If when the head is distending the perineum, the latter is well infiltrated with local anæsthetic and the mother given just enough trilene to take the edge off the contractions, very little additional doping will occur. If forceps application, or other complicated delivery becomes a necessity, a low spinal anæsthesia will not only cause no additional doping to the baby, but provide a salutary pelvic relaxation. The resultant baby may be damaged, but it will not be also doped.

Fortunately, the premature baby, being small, is born after a relatively short and painless labour during which the mother may have required little if any sedative. Infiltration of the perineum with local anæsthetic, and early and deep episiotomy, not only shorten the labour further but remove the pressure of the perineal muscles on the delicate and immature skull. The absence of doping may mean life or death to such a baby.

Those of us who employ so-called natural childbirth escape this problem of the doped baby. If the woman undertaking this method does require a sedative, it is in the bad half hour when the cervix is almost fully dilated, and by the time she is ready for delivery its effects have worn off. Since most of these cases are delivered with local anæsthetic in the perineum and episiotomy, no general anæsthetic is required; nor does the necessity for so-called prophylactic forceps seem to arise except in the rarest instances. As a result the baby cries the moment it is born, and makes an immediate and complete response to life that is very gratifying. It is my considered opinion that if this method of delivery were more widespread, a great deal of the resuscitation apparatus with which our delivery rooms are cluttered could be scrapped, or used on only the rarest occasions.

The forceps baby.—While the more difficult and dangerous (to the baby) forceps operations have yielded in many cases to the increasing safety of Cæsarean section, there seems to be a tendency to extend the use of so-called prophylactic forceps. What most of these forceps are prophylactic against I don't know, but being a private patient seems to affect their incidence. For instance, in the hospital in which I work, only about 5% of the ward patients are delivered by low forceps whereas 33% of the private patients are so delivered. Since the application of even this outlet type of forceps delivery carries with it the necessity of a general anæsthesia of some depth, the danger of doping is increased. Whether it is this extra doping or the pressure of the forceps on its head, this baby is more of a resuscitation problem than the one born spontaneously. Furthermore, it is the impression of those working in the nurseries that these babies are not as relaxed as those born spontaneously, and have more difficulty adjusting themselves to nursing.

It would be a fair statement that true prophylactic forceps are those applied to a head that is already bulging the perineum, and held there because of the perineum itself and/or the lack of power in the uterine contractions. The need for such forceps can be obviated in most cases by infiltrating the perineum with a local anæsthetic, doing an efficient episiotomy, and getting the woman to bear down with resolution. Of course, if she is deeply anæsthetised, or has had a caudal or spinal anæsthetic, she may have lost

the capacity to bear down, in which case forceps are prophylactic against the anæsthetic.

The baby that has been delivered by mid-forceps, especially if this has been associated with some rotating manœuvre, or the baby delivered as a breech, should be treated as a damaged baby. It may not have received damage, or the damage may not show up immediately, but until it is clearly out of the woods it should be given the benefit of the doubt, watched with the utmost vigilance, and handled with the greatest gentleness. The same is also true of the baby which, though born spontaneously, has had a long drawn-out labour, especially if the membranes have ruptured early. In the latter case it should be given penicillin.

The Cæsarean baby.—This baby may fall into any or all of the previously described problem categories. If the section was done for placenta prævia or toxæmia, it may be premature. If it followed a long, hard labour with ruptured membranes from the beginning, it may be damaged. If the mother had much sedative and the section was done under a general anæsthesia, it will almost certainly be doped. If the head is jammed tightly into the pelvis it may require a considerable pull with the forceps to dislodge it upwards. It is important that these factors be borne in mind.

If there is one thing more important than another in dealing with the Cæsarean baby, it is to drain it. Since I started to do this in the manner previously described (see Fig. 4) I have been amazed at the amount of amniotic fluid that will pour out and the length of time this continues. Even after holding such a baby upside-down for four minutes fluid will continue to drool from its mouth. It therefore stands in particular need of this drainage, which should be kept up for five minutes *by the clock*. Since a Cæsarean section is a major operation, the operator tends to be thinking as a surgeon rather than a pædiatrician. But unless he has cut or torn the uterine vessels there is nothing so urgent about the operation *per se* that cannot wait on this five minutes while the baby drains and the bulk of the placental blood flows into it. Such well-drained babies certainly do better and present fewer serious neonatal problems, nor do they seem to need to have their stomachs sucked out.

The problem of the Cæsarean baby will be lessened if the operation is done under local or low spinal rather than general anæsthesia. At least it will not be doped. I would suggest that we cease the stupid practice of getting rid of the Cæsarean baby by clamping its cord and handing it to a nurse the moment it is born; and give it the attention and respect due to it as a potential human being.

SUMMARY

The object of this paper has been to emphasize the importance of giving the newborn baby the utmost attention until it has made a complete and proper response to life. This means keeping it under constant surveillance for at least the first 10 minutes of its existence. It means handling it during that 10 minutes so that everything done to it counts towards its ultimate survival. It means that this responsibility must be undertaken by the obstetrician himself and not delegated to a nurse, intern or other less well qualified person.

MEDICAL WRITING

With approximately 10,000 medical journals and numerous pharmaceutical house publications available to the medical profession there must be a very large number of medical writers. During recent years there has been a tendency to publish vast amounts of data which in some cases, uses complicated formulæ and interpolations of tables and statistics. Some charts presented for publication are clear to the author, who is familiar with his own work, but to many readers little may be obtained from these figures. Authors preparing their papers should bear in mind that the general practitioner and specialists in other fields may not be familiar with complicated formulæ and statistical analyses; a tendency to more simplification in writing would be advantageous both to the author and reader. To the former it would mean more extensive reading and understanding of his work, and to the latter further application of the author's efforts. Work published must be accepted as it is read and not everyone is familiar with a slide-rule or the application of tables for x^2 probability scales. Confusion may arise from the use of mean, median, and mode; standard deviation, coefficient of correlation, coefficient of variation; and chi square test. Percentages should not be used when fewer than 100 observations are involved. Men and women should not be referred to as male and female, which might equally apply to herds of animals.

Medical writing should be carefully planned in advance, to avoid redundant expressions and to avoid confusion. Revisions should be numerous both by the authors and others to ensure an accurate interpretation of thought and findings. Tables should be easily understood and when ever possible should be presented as graphs with suitable scales and legends. If formulæ are used, their derivation should be included. If abbreviations are used, the full term should be included at the beginning of the article with the abbreviation following.

The following are recommended for reference to improve medical writing.

1. JORDAN, E. P. AND SHEPARD, W. C.: ℞ For Medical Writing, 1st ed., W. B. Saunders Co., Philadelphia, 1952.
2. BERNSTEIN, I. AND WEATHERALL, M.: Statistics For Medical and Other Biological Students, 1st ed., E. & S. Livingstone, 1952.
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FAMILY EPIDEMICS OF RINGWORM CONTRACTED FROM CATTLE

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LETENNEUR, a physician who practised medicine in the Vendée, France, was probably the first to give an accurate description of what the French call "dartres"—in Quebec they say "dattes"—a skin condition of animals transmissible to man.[4] He stated that he had proof from more than one hundred cases that herpes circinatus of man was being contracted from animals with "dartres." Letenneur blamed the dermatologists for not recognizing the etiology of this condition, on the grounds that they spent their time in the large cities and hospitals. This negligence was very surprising, he wrote, since the farmers themselves were quite aware of the contagiousness of the "dartres" and its transmission to man. He concluded his remonstrance with the following statement:

"Si les auteurs classiques n'en parlent pas, c'est qu'ils n'ont pas étudiés les maladies de la peau que dans les hôpitaux et dans les grands centres de population, et que souvent, dominés des idées préconçues, ils n'ont pas vu la vérité quand elle s'est montrée à eux. Je regarde donc comme un fait positif, et qui doit être aquis à la science, que l'herpès circiné et l'herpès tonsurans sont également contagieux, soit de l'homme à l'homme, soit des animaux à l'homme."

Last summer, when an opportunity to study such cases presented itself, we endeavoured to avoid the mistake which Letenneur had pointed out, and so the dermatologist and the mycologist went to the farms together to try and get a complete picture of the disease, its etiology and epidemiology.

The first farm visited was at Vankleek Hill, Ont. The owner had been treated at the Royal Victoria Hospital in May 1953 for a severe sycosis barbæ. Clinically he showed deep nodular suppurative lesions of the chin and submental area with heavy crusting (Fig. 1). There was a marked degree of adenopathy and secondary infection. Hairs taken from the infected areas showed large spores and hyphæ around the hair, the classical microscopical picture of a megaspore infection *sensu Sabouraud. Trichophyton discoides* Sabouraud, 1910, was isolated from the hairs. Two months later, his six year old son was brought to Montreal with a circular area of folliculitis on the head. The hair in this area was broken off close to the scalp. Wood's lamp examination was negative.[2] On direct microscopic examination the same features of large spores mixed with hyphæ surrounding the hairs were found. Again, *T. discoides* was found on culture.

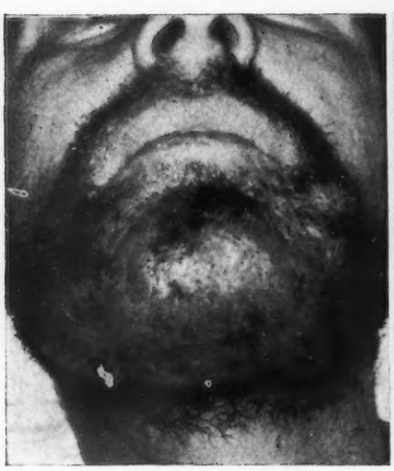

Fig. 1

Fig. 2

Fig. 3

In August when we visited the farm, all 14 cows were found to be infected with what is known in Quebec as "dattes." The infected areas showed little inflammatory response but the hair was broken off close to the skin as in the human cases. Hairs removed from these areas showed the same type of mycotic infection and cultures grew *T. discoides,* the commonest causative organism of ringworm in cattle in this area[3] as elsewhere.[1, 5] Examination of the other members of the family showed that the wife had lesions of a mycotic type on the right chest and right

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arm and another son aged ten had a lesion on the face from which *T. discoides* was isolated.

The second farm visited was near Lachute, Que. The farmer had been in the Royal Victoria Hospital in August 1953 for diagnosis and treatment of a deep folliculitis of the bearded area of the face (Fig. 2). Here we found 13 cows of which five were obviously infected. Specimens taken from the cattle grew *T. discoides*, the same dermatophyte grown from the farmer's beard. Examination of the family revealed that the two older sons, who helped with the milking, had areas of folliculitis on the forearms (Fig. 3). Direct examination and culture of these lesions showed that their infection was similar to that of their father and of the cattle.

The following day a farmer, his mother and his girl friend arrived at the clinic of the Royal Victoria Hospital from St. Eugène, Ont., eight miles from Lachute. The farmer had ringworm of the beard as well as lesions of the left chest and the neck which had persisted since April 1953. *T. discoides* was isolated from all these lesions. The mother had lesions of the right arm and under the breast which she believed she contracted from using the same towel as her son. The girl friend, unlike any of the other persons seen, did not live on a farm and had absolutely no contact with cattle. She had a circular area of folliculitis on her left forearm which grew *T. discoides*. It seems reasonable to assume that this was the result of putting her arm around the farmer's neck and represented direct contact from person to person.

The investigation of these epidemics corroborates the accuracy with which good observers described such epidemics without any knowledge of pathogenic fungi, more than one hundred years ago.

From a public health standpoint it would appear that veterinarians and physicians should work more closely together in endeavouring to control this condition, which appears to be on the increase in this area.

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THE EFFECT OF DIET ON THE METABOLIC ALTERATIONS OF PARAPLEGIA

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A MAJOR PROBLEM confronting those who supervise the care of paraplegics is the dietary; one of the problem's more controversial aspects, discussed more fully by the authors in a previous paper,[1] is the question of calcium and phosphorus intake. While low calcium diets have been recommended by some,[2] others [3] were unable to modify the high urinary calcium excretion in patients immobilized for fractures by raising or lowering the calcium intake. In view of the disagreement in the literature and the absence of relevant balance studies in paraplegia, it was decided to undertake a balance study in two such patients, using both high and low calcium and phosphorus diets, the minerals being added in the form of milk or of calcium lactate and sodium phosphate. The practical importance of such a study lies in the probability that the high urinary calcium levels regularly found in paraplegic patients are the result of the accompanying disuse osteoporosis and contribute considerably to the formation of urinary calculi, both renal and vesical, these being one of the major unsolved complications of paraplegia.

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METHOD OF STUDY

Basically, the study was divided into three 12-day periods, the first being a control period in which the analysed daily calcium intake was 0.95 gm. and the calculated phosphorus intake 2.2 gm. per day. During the second period there was low calcium (0.3 gm.) and low phosphorus (0.8 gm.) intake, while the third period of the study concerned high calcium (2.5 gm.) and high phosphorus (2.5 gm.) intake. The calcium over and above the control level was administered in the form of calcium lactate and phosphorus

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was added as monobasic sodium phosphate. In the study of the second patient (Y.C.), an additional 12-day period was included, the extra calcium and phosphorus being given as milk in place of the synthetic forms. Nitrogen intake was obtained by analysis, the figures varying somewhat in each subject and in each period. Daily nitrogen intakes were: first patient (G.P.)—13.5, 11.0 and 13.5 gm. respectively; second patient (Y.C.)—19.0, 11.0, 20.5 and 20.5 gm. respectively. Urinary calcium, phosphorus and nitrogen were determined in three-day pooled specimens, while six-day pooled stools were analysed for calcium. The details of these procedures will be found in an earlier paper.[4]

RESULTS

Figs. 1 and 2 show the results of the study. During the control period, both patients had the high urinary calcium levels expected in paraplegics who are immobilized. The first patient was excreting 300 to 400 mgm. calcium per day in the urine, the upper limit of normal being 200 mgm. per day; the second patient was losing 450 to 600 mgm. daily. During the period of low calcium intake, the urinary calcium loss was unaffected in either patient. With the high calcium intake, there was a rise in urinary calcium level by an average of 100 mgm. per day in G.P., while Y.C. showed no alteration in his urinary calcium excretion, either on a high milk

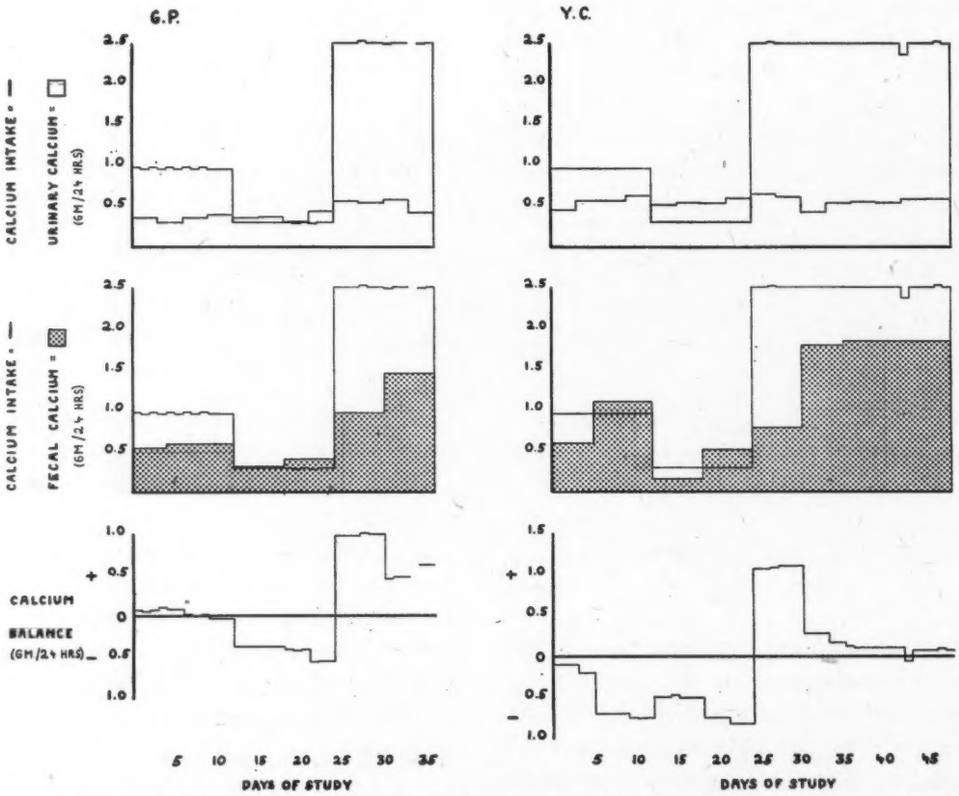

Fig. 1.—The effect of low and high calcium intakes on urine and fæcal calcium levels and on calcium balance in two paraplegic subjects.

SUBJECTS

Patient No. 1, G.P., a 24 year old man, was injured 40 days prior to study, sustaining a fracture-dislocation at D12-L1 with resultant flaccid paraplegia. Shortly after injury, an open reduction with tibial bone graft was carried out. Apart from several bouts of pyelonephritis and the finding of a positive Wassermann reaction in the blood, there were no complications.

Patient No. 2, Y.C., a 26 year old man, became paraplegic following injury to the back which resulted in a fracture-dislocation at L2-L3, two and a half months prior to study. Cystitis was present and a bladder calculus was discovered, being removed after the study. There was some recovery of movement on the left side. Paraplegia was of the flaccid variety, but there were frequent spasms of the left leg.

diet or when the calcium was given as lactate. Fig. 1 demonstrates clearly the close relationship between calcium intake and fæcal calcium level. The calcium balance shows that G.P. was in calcium equilibrium during the control period, but on a low calcium intake the balance became negative, later changing to a strongly positive balance on ingestion of large amounts of calcium. The second patient was in negative calcium balance during both the control and low calcium periods but was thrown into positive balance for

a short time when on the high intake, after which equilibrium was reached, in which state the patient remained for the rest of the study.

Fig. 2 demonstrates that the urinary phosphorus levels follow the intake fairly closely. The figures for nitrogen excretion in the urine showed that both patients were in nitrogen equilibrium throughout the study.

DISCUSSION

Since this study involved only two subjects, it would be misleading to draw too definite conclusions from the results; however, the authors believe that some generalizations might be inferred. Firstly, it appears fairly definite that a low calcium intake will not reduce the high urinary calcium level. This would confirm the findings of Howard[3] *et al.* Secondly, a high calcium intake is not necessarily accompanied by a rise in the urinary calcium; while in the first subject there was a rise of 100 mgm. per day on a high intake, the second subject showed no alteration. It is noted that patient Y.C. was in negative balance while G.P. was in positive balance during the control period. A low calcium intake would appear to predispose to a negative calcium balance, while a high calcium intake tends to keep the patient in positive balance, regardless of whether the patient was originally in positive or negative balance. There seems to be little doubt that a low phosphorus intake is accompanied by lower urinary phosphorus levels than is a high intake. The manner in which the added calcium or phosphorus was given did not appear to be of significance. The alterations in phosphorus and calcium intake had no significant effect on nitrogen metabolism.

CONCLUSIONS

From these studies, it is possible to infer that a high calcium, low phosphorus diet might be the diet of choice in paraplegia, since a high calcium intake increases the likelihood of keeping the patient in positive calcium balance without significantly altering the urinary calcium excretion. It would be wise to check the urinary calcium before and shortly after the institution of such a diet in each case, to be certain that no significant rise in excretion of calcium has occurred. Since two-thirds of urinary calculi in paraplegia are of the phosphate variety, it would seem advisable to keep urine phosphorus levels as low as possible; this could be achieved to a certain extent by reduction of the phosphorus intake. A high calcium, low phosphorus diet would involve severe milk restriction, the needed calcium being administered as lactate. Such a diet might aid in preventing the disuse osteoporosis seen in paraplegia and yet would not significantly affect urinary levels of calcium.

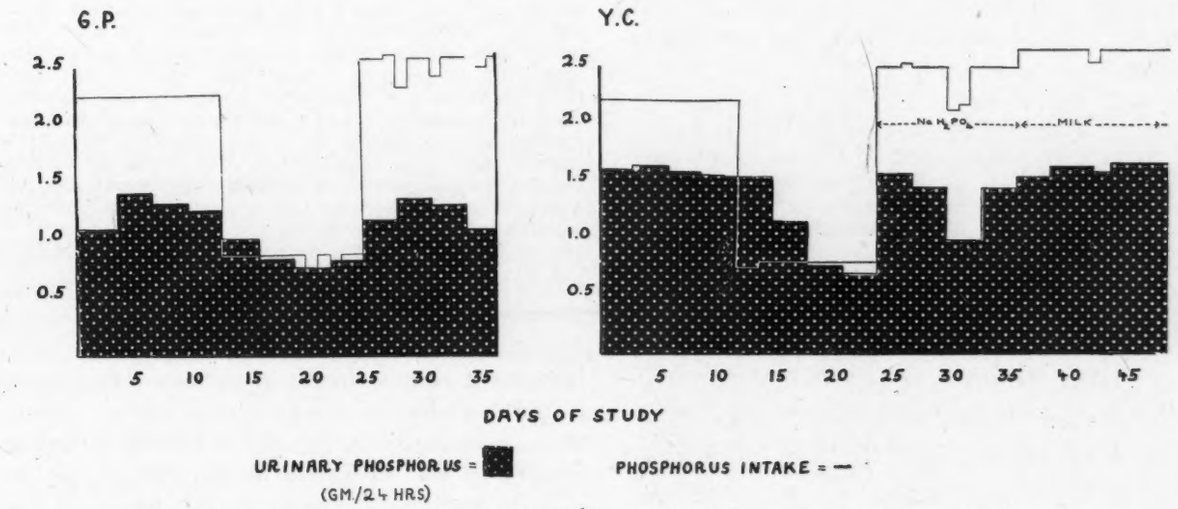

Fig. 2.—The effect of low and high phosphorus intakes on urine phosphorus levels in two paraplegic subjects.

SUMMARY

1. Two paraplegic patients were studied in regard to the effect of high and low calcium and phosphorus intakes on the excretion of these substances in the urine.

2. A low calcium intake did not lower the hypercalcinuria already present.

3. A high calcium intake raised the urinary calcium by 100 mgm. per day on the average in one patient, but did not affect the loss of calcium in the urine in the other case.

4. Low calcium intake affected the calcium balance adversely, while high calcium intake appeared beneficial in producing positive balance or at least equilibrium.

5. Lowered phosphorus intake was accompanied by a lowering in urinary phosphorus level.

6. It is concluded that a high calcium, low phosphorus diet might be the diet of choice in paraplegic patients who are immobilized. This study is being continued on a larger series of patients.

The authors wish to express their gratitude to Drs. J. S. L. Browne, G. Gringras and A. H. Neufeld for their invaluable aid, also to the nursing, dietetic and laboratory staffs of the Clinical Investigation Unit for their untiring effort, without which this work would not have been possible.

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RÉSUMÉ

Un des problèmes les plus importants du traitement des paraplégiques consiste à maintenir l'équilibre normal du calcium et du phosphore, et de prévenir l'ostéoporose et la formation des calculs urinaires. Afin de déterminer l'importance de l'apport de calcium dans le régime, les auteurs entreprirent de suivre de près 2 paraplégiques recevant un régime pauvre en calcium (0.3 grms.) et en phosphore (0.8 grms.). L'apport de ces deux minéraux fut par la suite augmenté au point d'en faire un régime riche en calcium (2.5 grms.) et en phosphore (2.5 grms.). La pauvreté de l'apport en calcium ne changea en rien l'excrétion urinaire, élevée chez ces malades immobilisés; par contre, l'ingestion de calcium en quantité supérieure à la normale produisit une élévation de l'excrétion d'environ 100 mgms. par jour, chez un malade. L'excrétion du phosphore suivit de près les variations du régime. Il semblerait donc qu'une diète riche en calcium et pauvre en phosphore soit indiquée chez les paraplégiques. Un tel régime signifierait une diminution considérable de la consommation du lait; le calcium nécessaire étant donné sous forme de lactate. On pourrait ainsi espérer réduire l'ostéoporose si fréquemment vue chez les paraplégiques, sans affecter grandement le niveau du calcium dans les urines.

M.R.D.

DISSEMINATED MONILIASIS WITH DEMONSTRATION OF THE ORGANISM IN THE BLOOD*

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DISSEMINATED MONILIASIS has been recognized only within recent years and Zimmerman (1950) has considered significant the absence of such infections prior to the generalized use of antibiotics in 1946. There is increasing evidence that the condition is developing secondarily to antibiotic therapy (Kligman 1952).

Disseminated moniliasis is to be differentiated from pulmonary moniliasis which, although a serious condition, does not inevitably result in death as does the disseminated disease. Moniliasis is the name given to infection with *Candida albicans, C. tropicalis, C. krusei,* and *C. parakrusei: C. albicans* is the best known of these because it causes thrush in infancy. The fungus is not pathogenic except under certain conditions: a decrease in the pH is known to favour growth of this organism in the infant's mouth and in the vagina. However, in almost all the cases of the disseminated disease reported, the infection has been a terminal one in patients suffering from some recognized severe debilitating disease: in these cases the use of antibiotics has prevented a terminal bacterial infection but has not relieved the underlying disease. The fungus has found conditions admirable for its rapid growth. It may have lain dormant in the tissues or been introduced from without, as in the case of possible introduction in intravenous glucose-saline reported by Duhig and Mead (1951).

Up to the present, clinical descriptions of the primary disease have been very few and have not allowed a clinical picture to be recognized. Gausewitz *et al.* (1951) gave perhaps the only full decription of disseminated moniliasis occurring as a primary disease in a boy aged 10 following prophylactic inoculation against diphtheria, pertussis and tetanus. He developed lymphadenopathy, hepatomegaly and splenomegaly

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and pulmonary infiltrations, but even this patient had oral thrush associated with penicillin and streptomycin therapy. Numerous writers have described monilial endocarditis (Wickler *et al.* 1942; Zimmerman, 1950; Wolfe and Henderson, 1951; Kunstadter *et al.* 1952). The organism has also been found in the cerebrospinal fluid, and autopsy has revealed the disseminated disease (Parillo, 1950).

The specific pathological finding has been a granulomatous reaction occurring in miliary foci in which the organism can sometimes be seen: the organism can also be recovered on culture from these foci. The granuloma resembles that seen in tuberculosis, disseminated histoplasmosis and coccidioidomycosis; but the organism is more often found lying free in the granulomatous or necrotic tissue, in distinction to the intracellular position of the other yeast-like organism, *Histoplasma capsulatum.*

It is considered that the organisms must travel by the blood stream to set up the disseminated foci but they have never been observed in the blood. *Histoplasma capsulatum* is the only fungus that has been identified in the white cells of the blood and therefore the finding of *C. albicans* within the white blood cells in this case is of interest.

CASE REPORT

A 33 year old Tamil man was admitted to hospital on January 2, 1953 complaining of cough and pain in the chest for the past 15 days. Two months previously he had been bitten by a rat or Indian bandicoot on the left big toe while asleep. After 10 days he developed rapid generalized œdema; he had dyspnœa and was severely ill, receiving treatment from a local doctor. He recovered "completely" at the end of three weeks although he still complained of feeling tired. There was no relevant past history.

On examination he was anæmic but there was no œdema. A grade 2 systolic murmur was heard maximally over the third left interspace adjacent to the sternum. He had a massive pleural effusion on the right side, and liver dullness extended down three fingerbreadths: the liver edge could not be felt, but there was tenderness over the liver area. The spleen was not enlarged to percussion. A small amount of ascites was present. The pulse was 50, regular and moderately full in volume. The blood pressure was 130/75. Radiography of the chest confirmed the presence of effusion but the heart size was normal.

The urine contained a slight amount of albumin and some red blood cells, but the urinary urobilinogen was not raised. The blood showed a hæmoglobin value of 58%, 2.8 million red cells, packed cell volume of 30%, mean corpuscular volume of 107.1 c.μ., mean corpuscular hæmoglobin of 34.3$\gamma\gamma$, mean corpuscular hæmoglobin percentage 32 and marked poikilocytosis. The total white cell count was 6,000, neutrophils 52%, lymphocytes 36%, monocytes 2%. The total plasma proteins were 7 gm. %, albumin 3 gm. %, globulin and fibrogen 4 gm. % but the formol-gel test was negative. Thymol turbidity 18 units, flocculation ++++. The serum van den Bergh showed only indirect bilirubin, 0.27 mgm. %. Examination of the myelo-erythroid layer from the hæmatocrit tube revealed many encapsulated oval bodies, constant in size and within the neutrophils; a few were seen within the monocytes. They measured 3-4 μ, stained a light blue with Leishman's stain, and had a central or peripheral chromatin mass. These were thought at first to be *Toxoplasma gondii* as the history of rat-bite was suggestive; but the shape, the presence of a halo and a negative serum dye-test for *T. gondii* (Beattie, 1953) ruled this diagnosis out. They did not resemble Leishman-Donovan bodies because there was no kinetoplast in addition to the trophonucleus. A diagnosis of *Histoplasma capsulatum* was considered from its known occurrence in the peripheral white cells.

The patient was running an irregular fever up to 101.5° F., from the day of admission. On the third hospital day he had macroscopic hæmaturia and he was given an alkaline mixture. Fundoscopy on that day was normal but an electrocardiogram showed heart block. A blood culture was taken and eight days later an aspergillus, which was considered a contaminant, was identified.

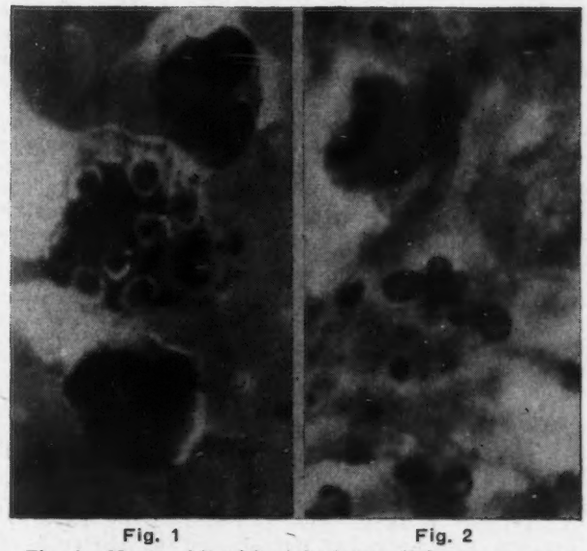

Fig. 1 Fig. 2

Fig. 1.—Neutrophil with eight intracellular yeast organisms. Stained Leishman's. **Fig. 2.**—Cluster of yeast organisms in granuloma of liver. Stained hæmatoxylin and eosin.

Following the microscopical identification of *H. capsulatum* 16 gm. aureomycin over 10 days was given in view of its value in systemic blastomycosis. However, there was no immediate change in his condition, the fever continuing and the anæmia increasing, although the urine became progressively free of red cells. Repeated search was made in the peripheral blood and sternal marrow for the parasites without success. As there was indefinite hepatomegaly, and neither splenomegaly nor generalized lymphadenopathy it was not possible to perform a puncture of these organs. The sternal punctures had shown a moderately cellular bone-marrow with normoblastic reaction.

A week after the cessation of aureomycin treatment a course of urea stibamine 2.5 gm. in divided doses intravenously and 163 gm. of sulphadiazine was given over 14 days: this resulted in a normal temperature for 10 days but afterwards the irregular fever returned. Pleural aspiration was carried out on three occasions with only partial success because of loculation. The fluid was very thick and yellow, but sterile on culture. Cultures of the sputum, urine and fæces revealed *C. albicans.* Repeated electrocardiograms showed no change in the tracings. Guinea-pig and mouse inoculations with the patient's blood were negative.

Following the false identification of the organism, potassium iodide had been given in gradually increasing doses with a maximum tolerated dose of 120 grains a

day. In addition iron and vitamin supplements were prescribed, together with regular intramuscular injections of crude liver extract. The anæmia continued to increase and ascites developed. He was placed on a low sodium diet and a mercurial diuretic given at regular intervals but with little response. Later, blood transfusions were given. Following these there was no response in the cell count or hæmoglobin and it was concluded that the marrow had become aplastic: unfortunately, no further marrow examination was made before death because of the patient's mental condition. A further serum van den Bergh test showed indirect bilirubin 0.3 mgm. %. Paracentesis abdominis was performed but the fluid rapidly reformed. The urinary output remained fair, but there were gradually increasing numbers of granular casts in the deposit.

On the eightieth day of admission terramycin orally was begun purely on empirical grounds. No observable response could however be demonstrated and the steady decline continued with the hæmoglobin falling to 20% and generalized œdema developing. Total proteins 6.0 gm. %, albumin 2.4 gm. % globulin 3.6 gm. %, thymol turbidity 20 units, flocculation ++++. He died on April 5, 93 days after admission.

Autopsy.—Autopsy, confined to removal of thoracic and abdominal organs, showed the following relevant findings: the subcutaneous tissues were very thin, slightly icteric and œdematous. The sternal and rib marrow was pale. The right pleura was greatly thickened and adherent to the thoracic wall. The lungs except for basal congestion showed no gross disease. The pericardium was not thickened but contained about three ounces of thin yellow fluid. The heart was slightly enlarged with the right ventricular wall thin and collapsed. On opening the heart, the ostia and the coronary vessels themselves were patent with no sclerosis.

On the surface of the right and left ventricular endocardium were small white plaques about three to four mm. in length, two or three in each ventricle. There was no evidence of myocardial disease. On close examination of the serous layer of pericardium over both ventricles, minute petechiæ could be seen.

The peritoneum contained about two pints of pale thin yellow fluid: the liver was not enlarged but was firm and the surface finely granulated with many small white pin-points resembling minute abscesses. The spleen was not enlarged, the lateral surface showing areas of perisplenitis and on section some prominent trabeculæ only. The gut was distended but showed no evidence of pathology beyond pallor. The pancreas was firm and rather gritty. The adrenals appeared quite normal though pale, but the kidneys were both large and very pale: the right kidney showed some subcapsular staining at both poles, and the left a few small petechiæ in the cortex.

Microscopic examination.—The heart showed some swelling of nuclei, lipochrome pigment deposition, and hæmosiderin scattered between the main bundles. There was slight thickening of the endocardium in the region of the white patches seen at autopsy, with very slight sub-endocardial cellular reaction but no organisms.

The lungs.—There was interstitial congestion and alveolar collapse.

The liver showed advanced portal cirrhosis with deposits of hæmosiderin and infiltration of the portal tracts by polymorphs and round cells. Granulomatous areas, with surrounding round cells, foreign body type giant cells and either a reticulated or caseous centre, were seen. In the periphery of the granuloma could be seen groups of yeast-like organisms, the wall staining well with hæmatoxylin but the rest of the cell poorly staining: some of these were to be seen within large mononuclear cells but not within the giant cells.

The spleen.—There were heavy fibrous trabeculæ, hæmosiderin deposition, reticulum cell proliferation, and granulomata together with yeast-like organisms.

The pancreas revealed interstitial fibrosis with areas of round cell infiltration, and many yeast-like cells, either singly or in clumps lying scattered about the inflammatory tissue. The islets were normal but few in number: the external secretory tissue showed swelling of the alveolar cells and hæmosiderin in the alveoli and ducts.

The kidney showed tubular necrosis, and the *adrenal* was of normal architecture, no organisms in either. However, the zona granulosa was filled with hæmosiderin, the rest of the cortex and the medulla to a lesser extent.

Bone-marrow.—Normoblastic reaction with many older polymorphs and eosinophils. Myelocytes common but blast cells comparatively scarce. Megakaryocytes well seen. No organisms seen in Leishman's or hæmatoxylin and eosin stained preparation.

The Prussian-blue reaction was strongly positive in the liver, spleen, pancreas and adrenal. Ziehl-Neelsen staining of all sections showed no acid-fast bacilli. The association of specific granulomata, yeast-like organisms, portal cirrhosis, heavy deposits of hæmosiderin in organs outside the liver and the bone-marrow findings suggested disseminated moniliasis, hæmochromatosis and maturation arrest aplastic anæmia.

MYCOLOGY

In this case we started with a prior idea of what might be found, in that the organism had been discovered inside the white blood cells and as far as was known *Histoplasma capsulatum* was the only fungus that had been observed in the peripheral blood.

At first it was thought that the organism grown on blood culture on two occasions was the pathogen, but following unsuccessful animal inoculations and the subsequent recognition of an aspergillus, it was realized that contamination had occurred. However, cultures grown from autopsy material (liver and spleen) under full aseptic precautions showed a spreading colony of white, smooth, faintly glistening growth at 37° C., at the end of 48 hours: subculture at 22° C. showed discrete smooth whitish colonies resembling *Staph. albus* on blood agar. The organism was subsequently found to be *C. albicans* because of the absence of the typical tuberculated chlamydospores of *H. capsulatum* in the mycelial growth and the formation of acid and gas in glucose, maltose and lævulose. These findings were confirmed by an experienced mycologist (Riddell, 1953).

DISCUSSION

The finding of intracellular organisms in the blood in this case resulted from a policy of examining the white-cell layer of the hæmatocrit tube in all cases of undetermined fever with a view to identifying hitherto unrecognized causal agents. In histoplasmosis, numerous observers have remarked that the presence of the organisms in the blood varies from day to day (Parsons and Zarofonetis, 1945) and the same may apply in disseminated moniliasis. In the case reported above they were identified only on one occa-

sion: it is important to exclude contamination because in the past many false reports have followed the discovery of contaminating organisms in blood films (Wenyon, 1926) but they have all been extracellular to the leucocytes; in the present case they were intracellular only.

When the patient came to hospital the history was difficult to interpret and the association of rat-bite with bizarre symptoms and signs could not be fully explained. It was realized that the rat-bite might be an unconnected occurrence, and the absence of the characteristic picture of rat-bite fever (*Spirillum minus* infection) supported this.

For the past two years we have been "fungus conscious" following the demonstration of a case of pulmonary moniliasis, another of cutaneous blastomycosis and one of torula meningoencephalitis (Roantree and Dunkerley, 1952). Similarly one of us (G.E.F.) had noted two chest radiographs of underground workers which suggested the atypical pulmonary calcifications described in histoplasmosis from the U.S.A. Middleton (1950) had suggested the possible occurrence of fungus disease in other arid areas of the world besides the southwestern states of the U.S.A.

The course of the anæmia showed it to be aplastic in type in the absence of hypersplenism or intravascular hæmolysis. However the peripheral blood changes did not truthfully indicate the functional state of the bone marrow, which both during life and at autopsy showed a cellular reaction: this is a recognized phenomenon in chronic aplastic anæmia, but usually in association with hypersplenism (Whitby and Britton, 1950).

The treatment carried out was empirical, and followed suggestions made by previous observers for histoplasmosis: neither aureomycin, terramycin, sulphadiazine nor urea stibamine had any effect, and in view of the subsequent finding of *C. albicans* this is not unexpected. Complete resistance to aureomycin and terramycin was shown by sensitivity tests on the organism cultured from autopsy tissues, in concentration of 10,000 μgm. of the antibiotic per ml. Potassium iodide was given but without observable effect.

SUMMARY

A case of disseminated moniliasis has been described in which the mycotic nature was recognized by finding the organisms in the white cells of the blood. This has not previously been reported in monilia infections. At post mortem the characteristic miliary granulomatous and necrotic lesions were found together with a well-developed hæmochromatosis.

This infection was recognized during the examination of the myelo-erythroid layer in the hæmatocrit tube after centrifuging.

We are grateful to Mr. B. Hari Shenoi, the late Senior Laboratory Technician, for his help in preparing the tissue sections; to Mr. Stan Klosevych for the photographs; to Professor C. P. Beattie of Sheffield, Dr. R. W. Riddell of the Brompton Hospital and Professor John Hamilton of Toronto for help. In addition, we would like to thank Dr. J. C. Jeffrey, Chief Medical Officer of the Kolar Gold Field, for permission to publish this report.

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OCULAR LESIONS IN GLANDULAR FEVER

The ocular manifestations of glandular fever are apparently not rare, and a recent author* has reviewed the literature and described a case of his own with iritis and papillœdema. The author divides lesions of the eye into two groups, first those due to direct involvement of the eye by the pathological lesion of infectious mononucleosis and second those effects on the eye produced indirectly by lesions of the central nervous system. In the former group are conjunctivitis, episcleritis, uveitis, optic neuritis and retinal œdema; while in the latter group are extraocular palsies and hemianopic defects.

The incidence of conjunctivitis varies with authors, some considering it to be of diagnostic importance. Of interest is the fact that puffiness of the eyelids is also not uncommon and may be as striking as in trichiniasis. Episcleritis and uveitis have also been described. Papillœdema and optic neuritis have been reported by some authors, and transient ocular signs due to involvement of the central nervous system are apparently not rare in glandular fever. The continuing awareness of this disease has added the ocular manifestations to those better known effects on the glands, meninges, blood and liver.

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MORTALITY FROM CANCER OF THE LUNG IN CANADA 1931-1952

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IN MANY COUNTRIES today the startling rise in the recorded death rate from cancer of the lung has made this a matter of public interest and concern. For example, Clemmesen[1] has reported that the crude mortality rate for this site of cancer among males in Denmark has increased from approximately 2.4 per 100,000 in 1931 to 16.5 in 1950. Dorn[2] has shown that, for white males in the United States, the age adjusted death rate per 100,000 for cancer of the lung and bronchus has increased from 3.7 in 1930-1932 to 18.5 in 1949, while Stocks[3] states that, in England and Wales, the male mortality as given by death certificates increased ten-fold in 22 years. This paper presents the experience in Canada in lung cancer deaths for the period 1931-1952 in order to assess any changes in mortality rates which may have occurred and to compare such changes with those reported in other countries.

It must be mentioned at the outset that it is not possible to combine the recorded numbers of deaths per year from cancer of the lung as given in the annual vital statistics reports for Canada. These deaths are coded according to the International List of Causes of Death but, during the period under review various revisions of this international classification have been made, thus making comparisons difficult. In the present instance the fourth, fifth and sixth revisions of the classification are involved. The variation in the categories to which deaths from lung cancer were assigned in these revisions are shown in Table I.

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In order to preserve comparability of mortality rates the Sixth Decennial Conference for Revision of the International List of Causes of Death recommended that, for the year 1949, all deaths in a country as a whole should be coded according to both the fifth and sixth revisions of the International List. This procedure showed the changes resulting from the application of the latest revision and provided a set of conversion factors by which data collected under the two revisions could be combined. Similar tabulations are not available for the fourth and fifth revisions of the International List, hence data on lung cancer deaths for the years 1931-1940 cannot be combined with those for subsequent years without qualification.

On the basis of these conversion factors the deaths in Canada from cancer of the lung have been reclassified for the years 1941 to 1949 to make them comparable with the years 1950 to 1952. The actual numbers of deaths shown by age-groups for males and females have been tabulated.* The years 1931-1940 include deaths classified in categories 47(b) and 47(d) of the fourth (1929) Revision of the International List; 1941-1949 include categories 47(b), 47(c), 47(d), 47(e) of the fifth (1939) Revision converted to

*The tabulated data have been omitted from the text to save space. The author should be consulted on matters involving them.

TABLE I.

VARIATIONS IN CATEGORIES COVERING LUNG CANCER DEATHS
Cancer of the respiratory system

Fourth revision (1929)	*Fifth revision (1939)*	*Sixth revision (1948)*
47 (a) *larynx	47 (a) *larynx	161 *larynx
47 (b) lung	47 (b) trachea	162 trachea, bronchus, lung (specified as primary)
47 (c) mediastinum	47 (c) bronchus	163 lung and bronchus (unspecified as to primary or secondary)
47 (d) other (bronchi, pleura, trachea)	47 (d) lung	164 mediastinum
	47 (e) pleura	165 thoracic organs (secondary)
	47 (f) mediastinum and unspecified	

*International List numbers.

TABLE II.

DEATHS FROM CANCER OF ALL SITES IN CANADA (1931 - 1952)

Year	*Total*	*Male*	*Female*	*Year*	*Total*	*Male*	*Female*
1931	9,578	4,744	4,384	1942	13,181	6,630	6,551
1932	10,024	4,939	5,085	1943	13,643	6,900	6,743
1933	10,653	5,143	5,510	1944	13,775	6,942	6,833
1934	10,581	5,239	5,342	1945	13,940	7,075	6,865
1935	11,156	5,450	5,706	1946	14,255	7,186	7,069
1936	11,694	5,706	5,988	1947	15,073	7,773	7,300
1937	11,963	5,969	5,994	1948	15,696	8,061	7,635
1938	12,038	5,901	6,137	1949	15,870	8,287	7,583
1939	12,399	6,246	6,153	1950	16,274	8,445	7,829
1940	13,322	6,793	6,529	1951	16,626	8,606	8,020
1941	12,952	6,538	6,414	1952	17,369	9,240	8,129

categories 162, 163 of the sixth (1948) Revision. It will be noted that all deaths from cancer of the lung not specified as secondary have been included in the years 1941-1952.

Deaths from cancer of all sites are shown by sex in Table II. The deaths for 1931 to 1940 include categories 45-53 of the fourth (1929) Revision of the International List; the years 1941-1949 include categories 45-55 of the fifth (1939) Revision* and the years 1950-1952 include categories 140-205 of the sixth (1948) Revision.* By means of the conversion factors mentioned previously, the deaths in 1941-1949 have been reclassified to make them comparable with the years 1950-1952.

From these data the proportion of all cancer deaths attributed to cancer of the lung may be calculated. These are shown in Table III.

Table III shows that, for both sexes, deaths from lung cancer have been reported with increasing frequency, rising from 2.2% of all cancer deaths in 1931 to 8.7% in 1952. This latter proportion agrees closely with the value 8.1% given by Dorn[2] for the United States. The increase for males as compared to females is to be noted. Whereas, in 1931, only 3.0% of all cancer deaths in males was attributed to lung cancer, in 1952 this had risen to 13.4%. In females the increase was much less pronounced, the figure rising from 1.4% to 3.2% over the same period.

In order to permit comparisons between age groups in any single year or between years the age-specific mortality rates have been calculated for each sex and for both sexes (see footnote on page 242). To indicate the age when death rates reach a maximum and to eliminate most of the sampling variability caused by having relatively small numbers of deaths in each age group for any single year, the age-specific mortality rates have been calculated also for three three-year periods, 1931-1933, 1941-1943 and 1950-1952. These data are shown in Table IV. It will be noted that, for males, the highest rate for the

TABLE III.

PROPORTION OF ALL CANCER DEATHS ATTRIBUTED TO LUNG CANCER

Year	Percentage		
	Total	Male	Female
1931	2.2	3.0	1.4
1932	2.1	3.0	1.3
1933	2.5	3.4	1.7
1934	2.5	3.3	1.6
1935	2.8	4.0	1.7
1936	3.0	4.1	1.9
1937	3.2	4.5	1.8
1938	3.3	4.6	2.0
1939	3.7	5.6	1.7
1940	4.0	5.9	2.0
1941	4.2	6.2	2.2
1942	4.1	6.1	2.1
1943	4.6	6.6	2.5
1944	4.7	6.7	2.6
1945	5.0	7.5	2.4
1946	5.5	8.5	2.4
1947	6.0	9.3	2.6
1948	6.2	9.7	2.6
1949	7.2	10.8	3.3
1950	7.5	12.2	2.5
1951	8.0	12.7	2.9
1952	8.7	13.4	3.2

TABLE IV.

AGE-SPECIFIC DEATH RATES PER 100,000 POPULATION FOR CANCER OF THE LUNG IN CANADA (1931 - 1952)

Male

Year	All ages*	Under 30	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85+
1931 - 1933	2.8	0.2	0.9	2.0	3.0	5.2	8.2	12.7	12.5	15.1	10.7	15.8	8.2	11.4
1941 - 1943	7.1	0.3	0.9	3.2	5.4	12.5	18.9	27.0	33.7	34.5	30.6	30.0	27.9	14.3
1950 - 1952	15.8	1.0	0.8	3.0	6.5	16.7	37.2	59.7	77.8	102.9	86.3	83.9	59.7	71.0

Female

Year	All ages*	Under 30	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85+
1931 - 1933	1.5	0.2	0.6	0.9	1.6	2.4	2.6	6.1	8.3	9.8	9.8	7.8	5.2	2.3
1941 - 1943	2.6	0.2	0.8	1.5	2.3	5.0	4.8	8.7	12.4	13.8	15.6	12.8	11.3	14.8
1950 - 1952	3.3	0.1	0.6	1.1	2.2	3.3	5.9	8.0	13.8	18.7	21.6	30.9	29.9	23.3

*Crude Mortality rate.

*Excluding *Hodgkin's Disease* (categories 44b of the fifth and 201 of the sixth Revisions of the International List) and *Leukæmia* and *Aleukæmia* (categories 74 of the fifth and 204 of the sixth Revisions of the International List).

periods 1941-1943 and 1950-1952 occurred at 65-69 years of age. For females the highest rate in the period 1941-1943 occurred at 70-74 years of age and in the 1950-1952 period at 75-79 years of age.

Variations in the annual deaths from cancer of the lung at all ages can be compared by means of standardized mortality rates. These rates are shown in Table V and have been standardized on the 1951 census population of Canada.

It will be noted from Table V that the sex ratio of lung cancer deaths has increased steadily from 1.9 to 1 in 1931 to 4.9 to 1 in 1952. This latter value agrees with those reported by both Dorn[2] and Clemmesen.[1]

The standardized death rates offer a measure of the increase in deaths from lung cancer as shown in Table VI. It will be noted that the two periods 1931-1940 and 1941-1952, have been treated separately since, as already mentioned, the data for these periods are not truly comparable. The increase for the entire period has been shown also, but it must be accepted with certain reservations.

TABLE V.

STANDARDIZED DEATH RATES PER 100,000 POPULATION FOR CANCER OF THE LUNG IN CANADA (1931 - 1952)

Year	*Total*	*Male*	*Female*	*Sex-ratio*	*Year*	*Total*	*Male*	*Female*	*Sex-ratio*
1931	2.4	3.0	1.6	1.9	1942	4.8	7.0	2.5	2.8
1932	2.4	3.3	1.6	2.1	1943	5.5	7.9	3.0	2.6
1933	2.9	3.6	2.1	1.7	1944	5.6	7.9	3.1	2.5
1934	2.8	3.6	1.9	1.9	1945	5.9	8.8	2.9	3.1
1935	3.4	4.5	2.1	2.1	1946	6.6	10.2	2.9	3.5
1936	3.6	4.7	2.4	1.9	1947	7.5	11.7	3.1	3.8
1937	3.8	5.1	2.3	2.2	1948	7.8	12.3	3.2	3.8
1938	3.8	5.2	2.5	2.1	1949	8.8	13.6	3.8	3.6
1939	4.4	6.5	2.1	3.1	1950	9.2	15.3	2.9	5.2
1940	4.9	7.2	2.4	3.0	1951	9.5	15.4	3.4	4.5
1941	5.0	7.3	2.5	2.9	1952	10.7	17.0	3.7	4.9

TABLE VI.

INCREASE IN DEATHS FROM CANCER OF THE LUNG IN CANADA

Between the years	*Number of times deaths have increased*		
	Total	*Male*	*Female*
1931 - 1940	2.0	2.4	1.5
1941 - 1952	2.1	2.3	1.5
1931 - 1952	4.5	5.7	2.3

SUMMARY

An analysis has been made of the deaths in Canada from lung cancer for the years 1931-1952. The data for the years 1941-1952 have been made comparable by means of conversion factors and include all deaths attributed to lung cancer not specified as secondary.

In 1931 deaths reported as due to lung cancer represented 2.2% of all cancer deaths. This figure had increased to 8.7% in 1952. For males the increase was from 3.0 to 13.4% and for females from 1.4 to 3.2%.

The highest mortality rate for lung cancer in any age group of men in the period 1931-1933 was 15.8 per 100,000. This had increased to 34.5 in 1941-1943 and to 102.9 in 1950-1952. Among females the age-specific mortality rates for lung cancer were considerably lower but increased from a maximum of 9.8 per 100,000 population in 1931-1933 to 15.6 in 1941-1943 and to 30.9 in 1950-1952. The maximum mortality rate for males in each of the periods 1941-1943 and 1950-1952 occurred at 65-69 years of age. For females the maximum rate, for the period 1941-1943 occurred at 70-74 years of age, and for the period 1950-1952 at 75-79 years of age.

When corrections were made for changes in the age structure of the population, it was found that the death rates had increased from 2.4 per 100,000 population in 1931 to 10.7 in 1952. For males the standardized death rate has increased from 3.0 in 1931 to 17.0 in 1952 and for females from 1.6 in 1931 to 3.7 in 1952.

The ratio of male to female deaths from lung cancer has increased from 1.9 to 1 in 1931 to 4.9 to 1 in 1952.

The author wishes to acknowledge the assistance of the Dominion Bureau of Statistics in this study.

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ADDITIONAL BOTULISM EPISODES IN CANADA*

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SINCE THE PUBLICATION about a year ago of a report on human botulism in Canada,[1] four additional episodes have come to light, and will form the subject of this communication. First to be described is a previously unrecorded outbreak in Ontario, in 1934, attributed to home-bottled venison, and involving two persons, one of whom died. An account will then be given of three other outbreaks which occurred towards the end of 1953 in a relatively small area of the southern interior of British Columbia. The foodstuffs implicated were home-bottled spinach, beets, and corn on the cob, respectively. Samples of corn, responsible for two fatalities at Grand Forks yielded a highly toxigenic strain of *Clostridium botulinum* type A. Another toxigenic type A strain was isolated from two jars of bottled spinach, after a companion jar had apparently caused mild botulism in two persons at Rock Creek. Soil samples from these vicinities were shown to contain similar organisms. Finally, home-preserved beets were probably responsible for a single case of moderately severe botulism at Gilpin; but in this instance no samples were available for bacteriological examination.

CASE REPORTS:

Two cases at Corbetton, Ontario, in 1934.

[The author is indebted to Dr. R. B. Kerr, Professor of Medicine, University of British Columbia, for drawing his attention to this episode, and to Dr. H. H. Hyland, Associate Professor of Medicine, University of Toronto, for supplying details on which the following account is based.]

A man aged 35, and his wife aged 26, both of Hungarian birth, were admitted to the Toronto General Hospital on October 30, 1934. Their home was in the village of Corbetton, Dufferin County, not far from Orangeville, Ont. Five days before, they had developed severe abdominal pain, vomiting, dizziness, diplopia, dysphagia and constipation.

On admission, the man was totally unable to swallow, owing to paralysis of the pharynx and palate. His pupils were fixed, the external rectus muscles were weak, and there was paralysis of the left diaphragm and bladder. He vomited repeatedly. His temperature was slightly raised (up to 100° F.), but the cerebrospinal fluid showed no abnormality. He died on November 2, the third day after admission to hospital, and the eighth day after the onset of illness. The only significant post mortem findings were œdema and hyperæmia of the brain, acute gastro-enteritis, and acute cystitis.

The woman's symptoms, though similar, were of more gradual onset and less severe. She recovered progressively over a period of several weeks.

The wife stated that on October 4, three weeks before their illness began, her husband shot a deer through the abdomen, and left it alive overnight. Next day, he found it dead, skinned it, and cut away the meat, giving some to neighbours, and putting the rest in the cellar for a couple of days. On October 7, this meat was boiled in a pot for three or four hours, then transferred while hot to quart jars (also boiled). Rubber rings and glass sealers were fitted to the tops of the jars, and the lids screwed on. This preserved meat was eaten between October 8 and 25, some jars being given to neighbours, none of whom became ill. The victims opened their last jar, and consumed its contents, on the day before symptoms developed. Although no samples of the meat could be procured for bacteriological examination, the clinical and other data were considered to justify the diagnosis of botulism.

Two Fatal Cases at Grand Forks, B.C., 1953.

[The following episode occurred within the boundaries of the West Kootenay Health Unit, whose Medical Health Officer, Dr. A. F. Balkany, conducted a careful enquiry, and kindly procured much of the information incorporated in this outline.]

On December 22, 1953, at Grand Forks, B.C., a Doukhobor farmer and his seven year old daughter ate a small quantity of home-preserved corn on the cob at the evening meal. The wife and two younger sons had none of the corn. About 36 hours later, the man began to vomit, felt weak, complained of seeing double, and developed a staggering gait, as though inebriated. Next day, the girl was similarly affected. The local physician, Dr. D. A. Perley, was called. Suspecting botulism, he urged removal of both patients to the nearest hospital equipped with a respirator, since there was obvious dyspnœa.

They were admitted to the Trail-Tadanac Hospital, under the care of Dr. J. S. Daly, on December 26. At this time, the man's face was flushed, his pupils dilated, and speech slurring. There was weakness of the muscles of the face, neck and shoulders (his head falling back on being lifted), and reflexes generally were feeble. He was restless and complained of intense headache. However, a specimen of cerebrospinal fluid, sent to the Provincial Laboratories at Vancouver, showed no abnormality. His temperature was likewise normal. Botulism was diagnosed, and efforts were made unsuccessfully to procure botulinus antitoxin for specific therapy. The patient was placed in an oxygen tent, but his restlessness and weakness increased, and he died on December 28, apparently from heart failure.

The symptomatology and course of the illness in the child were similar. She seemed to improve somewhat in the respirator, judging by her colour and pulse, but after 36 hours, her condition rapidly deteriorated. Her temperature rose to 103° F. and pulse rate to 160, and on December 29 she died, the terminal events again suggesting cardiac rather than respiratory paralysis.

The corn had been harvested three or four months before from a patch of land previously used as a cow pasture.* The family had already consumed several bottles of this crop without harm. The housewife stated that when putting up the corn, she had boiled it in the bottles continuously for four and a half hours; and before placing the corn from the implicated bottle on the table, had reboiled it for "several minutes." The man ate the corn from at most half a cob, while the girl took

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*The previous year, corn had been grown on another patch some 100 yards away. Some of this crop was bottled off the cob. After a sample was fed to the chickens, five died of typical limberneck.

only one or two small bites. The remaining cobs were thrown out into the snow, whence they were retrieved after 15 days by Dr. Perley, and forwarded by him to the author.

On arrival in Vancouver three days later, the consignment of corn, comprising three and a half soggy but still intact cobs, with a sickly sweet, fermented odour, was at once examined. About 5 gm. of kernels from each cob was macerated in roughly 20 c.c. of sterile, physiological saline. Smears made from these suspensions showed under the microscope large numbers of Gram-positive cocci, and also some round-ended Gram-positive bacilli, the latter mostly embedded in fibrils of corn. The suspensions were centrifuged, and various amounts of the supernatants injected into mice intraperitoneally. All showed the presence of a heat-labile lethal factor, the most potent suspension containing roughly 200 mouse M.L.D. per c.c. By injecting mice with mixtures of several M.L.D. of the suspension plus 0.1 c.c. of types A, B and E antitoxin, respectively, the lethal factor was shown to be type A botulinus toxin.

Large tubes of beef infusion-peptone-broth with added ground meat, were brought near to boiling point in a water bath, and 3-4 gm. amounts of corn kernels were introduced. The inoculated tubes were held for a few minutes at around 90° C. before being allowed to cool, and were then incubated anaerobically in a McIntosh-Fildes jar for four days at 37° C. Two of these cultures were found to contain type A botulinus toxin of potency around 20,000 mouse M.L.D. per c.c. Loopfuls of these cultures, streaked on to beef infusion agar containing 10% of human blood, yielded on overnight inoculation at 37° C., a slightly hæmolytic, spreading growth of a Gram-positive sporulating bacillus. Without special difficulty, a pure culture was isolated having the biochemical and toxigenic properties of *Clostridium botulinum* type A.* Subsequently, selected mutants of this strain, grown in the above-mentioned ground meat medium with 2% of added dextrose, produced type A toxin of potency between 300,000 and 1,000,000 mouse M.L.D. per c.c. Certain of these mutants displayed lytic phenomena suggestive of bacteriophage action.

On May 14, 1954, nearly six months after the fatalities, four samples of soil were collected from the farm by Dr. Balkany. Grab samples were taken from two areas about 10 yards apart in both the 1952 and the 1953 corn patches. In all four soil samples the presence of a toxigenic strain of *Cl. botulinum* type A was demonstrated.

Two Mild Cases at Rock Creek, B.C., 1953.

On November 17, 1953, in a large farm house at Rock Creek, about 25 miles due west of Grand Forks, B.C., three adults and a young child ate an evening dinner of roast beef, potatoes, Yorkshire pudding and home-preserved spinach. The group consisted of the rancher's wife, aged 56, her daughter, aged 26, the son-in-law, and a four and a half year old granddaughter. The younger woman, being especially fond of spinach, consumed a rather large quantity of it: the others ate moderate amounts. The rancher (of Welsh origin) was not present.

Next day, the older woman developed painful abdominal distension with constipation. These were her only symptoms, but they persisted for about six weeks. The younger woman became more definitely ill. About 36 hours after eating the spinach, she felt dizzy and queer on arising, and could not focus her eyes on objects. The following day, she complained of general malaise and weakness, difficulty in swallowing, and persistent visual trouble. She was unable to judge distances; e.g., when crossing the street she could not estimate the distance of approaching cars. She consulted an optometrist, but the spectacles prescribed did not alleviate her ocular symptoms. On November 25, she noticed marked weakness in her hands and wrists, especially when wringing out clothes or picking up her little girl. On the 28th, the swallowing difficulty was such that food had to be washed down at mealtimes with fluid. As these symptoms persisted, on December 14 she visited Dr. J. J. Gibson in Penticton, who found her reflexes normal, while urinalysis, cerebrospinal fluid examination, and blood serological tests were reported negative. A tentative diagnosis of influenza was made, but the patient was told to report for a re-check in a few days if her condition did not improve.

On December 22 the patient returned, still complaining of visual focusing difficulty, slight dysphagia, weakness in both arms, marked general malaise and depression. On this occasion, she alluded to the deaths of some 30 chickens on the farm about a month before. On November 21, the chickens had been fed a mash mixed with spinach. (The spinach had been thrown into the slop-pail when the first jar opened for the family's evening meal on November 17 showed soft and gassy contents.) Within 14 hours, 16 chickens had died. During the next 24 hours, another 14 chickens died, while three which displayed advanced limberneck were destroyed. On hearing this story, Dr. Gibson reported the case as food poisoning to Dr. D. A. Clarke, Director, South Okanagan Health Unit, who considered the episode a possible example of botulism, and promptly made a careful investigation.

The patient's physical disabilities continued for about 10 weeks, while for several weeks thereafter she lacked initiative and felt depressed to the extent of "sitting around the stove most of the day." However, when visited by Dr. Clarke on April 26, 1954, she appeared in excellent physical and mental health, and had discarded her spectacles.

(Incidentally, the rancher himself reported having a bout of diplopia two summers before. On June 14, 1951, while plowing a field, "he looked up at the sun," saw double, and drove his team of horses through a wire fence. He consulted Dr. Gibson about his visual disorder, and was referred by him to Dr. P. Found of Kelowna. The patient clearly recalls that on his way to the city, every object had two images. On examination, his visual fields and fundi proved normal, and a toxic paralysis of the left superior rectus muscle was diagnosed. The diplopia lasted for two weeks, then completely disappeared, and has not recurred. In retrospect, no particular foodstuff could be implicated, but the rancher stated he probably consumed several home-preserved vegetables around that time.)

The suspected spinach had been put up by the cold pack process early in August 1953. After being shrunk by heat for a few minutes, it was transferred to 24 oz. and 16 oz. jars with metal lids and screw-on-tops, water was added, and the jars were then boiled for two hours. They had been stored in a cool cellar for about three and a half months before the evening meal in question, when the contents of one jar were emptied into a glass casserole. A little water and butter were added, the covered dish was heated for about 10 minutes, and the spinach was served as already described.

The foregoing information was kindly supplied by Dr. Clarke, who also procured for laboratory examination six jars of spinach and two samples of the well-manured, black garden soil in which it had been grown. The jars were firmly sealed, and their contents appeared normal in colour, consistency and odour. The pH of the spinach juice was about 6.2. Juice from each jar, injected in 0.3 c.c. amounts into mice, proved harmless. By techniques similar to those employed with the corn samples, *Cl. botulinum* type A was isolated from each of two 24 oz. jars of spinach. One of the cultures proved at first atoxic, but eventually gave rise to a toxigenic mutant, which yielded toxin containing approximately 30,000 mouse M.L.D. per c.c. The other culture produced potent toxin on initial isolation, its titre being at least 100,000 mouse M.L.D. per c.c. Both soil samples showed the presence of a toxigenic type A strain.

*In September 1953, at the Sixth International Microbiological Congress, meeting in Rome, the Subcommittee on Classification of Anaerobes recommended discarding the designation *"parabotulinum"* for types A and B of this species.

Suspected Case at Gilpin, B.C.

A Doukhobor woman residing at Gilpin, a small community about seven miles east of Grand Forks, B.C., was admitted to St. Luke's Hospital, Spokane, Washington, on December 22, 1953. She was suffering from headache, inability to see clearly or to speak plainly, dysphagia, and weakness of arms, legs and neck. Fever was at no time observed, and the cerebrospinal fluid taken on admission showed no abnormality. Dr. D. A. Perley of Grand Forks, who first saw the patient, considered her illness to be botulism, but the hospital diagnosis was poliomyelitis. The patient made a good recovery, and after 18 days under observation, was discharged without residual weakness or other sequelæ.

Symptoms had developed three days after the woman and her family had eaten home-preserved beets. These beets had been sealed in jars after being scalded for 10 minutes in boiling water. The jars were then stood in boiling water for one hour. The patient claimed to have heated the contents of one jar for half an hour before serving them to the whole family. Nobody else became ill, and other jars of beets were eaten subsequently without evident harm. However, the contents of two other jars were thrown away, because they appeared "overcooked." Unfortunately, no beet samples remained for laboratory examination. Dr. A. F. Balkany, Director of the West Kootenay Health Unit, again furnished most of the data on this case.

DISCUSSION

The known occurrences of proven or suspected botulism in Canada have been summarized in Table I. The revised list includes 14 episodes, involving 63 persons, with 35 deaths—a case fatality rate of 55.5%. The peculiar distribution of the disease in this country, alluded to previously,[1] is accentuated by the present report. One-half of all recorded outbreaks have occurred in Southern British Columbia. Moreover, since 1948, within a 25-mile radius of Grand Forks, B.C., there have been three proven type A episodes, affecting six persons, of whom four died. By contrast, botulism has been noted only twice east of the Great Lakes, both instances being in southern Ontario, about one year and 130 miles apart.

This distribution reflects on a small scale the situation in the United States, where Meyer and Eddie's careful records[2] reveal that California and other states west of the Rocky Mountains have the highest prevalence of botulism, particularly of the type A variety. Further, the presence of type A cultures in the botulogenic soils of an asparagus bed,[3] two corn patches, and a spinach garden, all in the Grand Forks area, strengthen the suggestion made in an earlier communication[1] that the far westerly regions of the United States and Canada share a comparatively heavy telluric incidence of type A spores.

Other factors clearly conducive to botulism are a warm climate, abundance of vegetable produce, and faulty home-bottling customs. These conditions are all fulfilled in the Grand Forks area, where the rich soil yields abundant crops, the prevailing summer temperatures may make it difficult to store preserves in a cool place, and Doukhobor farm communities observe generally low hygienic standards. In such an area, it is not surprising that spoilage of home-preserved vegetables should be very common; that limberneck among chicken flocks should quite frequently occur; and that local physicians should recall occasional human cases of undiagnosed paralyses of possible botulinic origin.

In these circumstances, the *prevention* of further outbreaks of botulism is primarily a public health educational problem. Four well-established control measures need especially to be stressed. First, vegetables and fruits for home-

TABLE I.

HUMAN BOTULISM IN CANADA

Year	*Cases*	*Deaths*	*Place of occurrence*	*Foodstuff implicated*	*Type*
1919	23	12	Dawson City, Y.T.	Commercially canned beets	
1933	3	1	Zurich, Ont.	Home-canned tomatoes	
1934	2	1	Corbetton, Ont.	Home-bottled venison	
1940	5	3	Whitehorse, Y.T.	Uncooked salmon eggs	
1941	5	2	Maple Creek, Sask.	Home-canned fish	
1944	3	3	Nanaimo, B.C.	Home-canned salmon	E
1945	8	7	Markham Bay, N.W.T.	Uncooked seal meat	
1948	2	2	Grand Forks, B.C.	Home-bottled asparagus	A
1949	4	0	Masefield, Sask.	Home-canned beef	
1949	2	1	Vancouver, B.C.	Home-pickled herring	E
1952	1	1	Natal, B.C.	Home-pickled trout	E
1953	2	0	Bridesville, B.C.	Home-bottled spinach	A
1953	1	0	Gilpin, B.C.	Home-bottled beets	
1953	2	2	Grand Forks, B.C.	Home-bottled corn-on-cob	A

Total cases 63; total deaths 35; case fatality rate 56 per cent.

preserving must be thoroughly washed and free from blemishes. (The corn which caused the Grand Forks fatalities showed cankerous spots). Secondly, proper containers should be used, preferably special glass sealers with tight-fitting tops and screw-on lids. It is not only false economy, but a menace to health, to put up small quantities of asparagus, peas and string beans in 6 oz. mayonnaise jars, such as were received (showing evidence of infection) from the farm where the lethal corn was grown.

Thirdly, the failure of even prolonged boiling to destroy type A botulinus spores is still not appreciated by many intelligent housewives, whose techniques of bottling may be otherwise impeccable. The Grand Forks bottled corn was allegedly boiled for four and a half hours: the Rock Creek spinach for two hours. Yet the corn was heavily polluted with type A organisms and toxin, while type A organisms were isolated from two out of six jars of the spinach, after two other jars had been demonstrated epidemiologically to be toxic. Over 30 years ago, Koser, Edmondson and Giltner,[4] and Schoenholz, Esty and Meyer,[5] carefully investigated the conditions promoting toxin production in commercially canned spinach, and showed that this product was very liable to cause botulism when harvested from fields heavily infected with *Cl. botulinum*, then insufficiently cleaned, and finally heated only to 100° C. for three hours. These observations were taken to heart by commercial canners, largely as a result of the thorough studies and strenuous campaigns of Meyer.[6] But among home processors the lesson needs to be far more effectively disseminated that to ensure sterilization of spinach in quart sealers, exposure to 250° F. (121° C.) for 70 minutes in a steam pressure cooker is necessary. Corn is still more difficult to sterilize, and requires not less than 80 minutes exposure to similar conditions.

Fourthly, most deaths from botulism could be avoided by the simple precaution of thoroughly heating home-bottled foodstuffs to 100° C. for 10 to 15 minutes, thus destroying any of the relatively heat-labile, preformed toxin. But the episodes described above illustrate that a mere warming up of a mass of spinach, or even the immersion for several minutes in boiling water of a poor heat-conductor like corn on the cob, may only detoxicate the surface portions and be an insufficient safeguard.

The *diagnosis* of an outbreak of acute botulism, such as occurred in 1934 at Corbetton, Ont., and at Grand Forks, B.C. in 1948 and 1953, should not confuse the alert clinician. An isolated subacute case may present more difficulties, and the brief account of the patient from Gilpin was inserted mainly as a reminder that this condition is apt to be mistaken for poliomyelitis. The ambulatory cases at Rock Creek, which could so easily have been overlooked, illustrate the likelihood that mild botulism occasionally masquerades as influenza, neurasthenia, or "a touch of polio," and indeed may be endured without benefit of medical opinion.

For many years in Germany, and more recently in France, these lesser manifestations of botulism have been well recognized—a fact which no doubt partly accounts for the relatively low case fatality rates in those countries. In 1948, Wasmuth[7] reported that inclusive statistics for human botulism in Germany showed an average fatality rate of about 15%; while Legroux, Levaditi and Jéramec[8] claimed the fatality rate among over 1,000 cases during the occupation of France was less than 2%. Admittedly, the contrast between these figures and the 55 to 65% fatality rate ascribed to botulism in North America can hardly be due solely to greater familiarity in Western Europe with the more occult forms of the disease. (In Germany and France, type B intoxication has predominated, while the foodstuffs characteristically involved there have been prepared meats, mostly porcine.) Nevertheless there is insufficient realization on this continent of the possible botulinic significance of syndromes in which constipation and abdominal discomfort, or inability to swallow freely, may be associated with double vision or difficulty in visual accommodation, with such ancillary complaints as dryness of the mouth, dizziness or ready fatiguability.

The *treatment* of botulism is not viewed so pessimistically in some European countries as in North America. French authorities[8] claim good results from specific therapy, and recommend a combination of antitoxin and toxoid injections. Until the toxin type involved in a given case or outbreak is determined, this would logically entail administration of polyvalent types A, B and E antitoxin and toxoid. No such products are at present manufactured in Canada or the United States. Symptomatic treatment in severe cases should include use of a mechanical respirator, with oxygen; careful attention to clearing the

pharynx and bronchi; feeding by duodenal tube; and fluids parenterally. Pederson and Christensen[9] have stressed the importance of treating the state of shock which accompanies acute botulism and advocate transfusions of blood and dextran. Exsanguination to the extent of 1 litre with immediate replacement by plasma has also some theoretical merit based on the experimental demonstration of botulinus toxin in the blood stream before its elimination in part by the kidney. Further absorption of toxin should of course be prevented and its elimination encouraged as far as possible at the outset by gastric lavage and high enemas.

SUMMARY

1. Four previously unrecorded botulism episodes in Canada are described. These bring the total known outbreaks in this country to 14, with 63 persons involved, 35 deaths, and a case fatality rate of 56%. Seven outbreaks have occurred in British Columbia in the last 10 years, 13 persons being affected, nine fatally.

2. One outbreak, due to venison, occurred in southwestern Ontario in 1934. Two adults were affected, one fatally. Three other outbreaks occured late in 1953 in the southern interior of British Columbia, in or near Grand Forks. Corn on the cob was responsible for the death of a young girl and her father; spinach caused mild botulism in two adults; and beets were the probable vehicle of moderately severe botulism in another instance. All these products were home-bottled.

3. In the recent outbreaks, toxigenic strains of *Cl. botulinum* type A were isolated from the residual corn, and from two out of six companion jars of spinach. Similar organisms were present in the soil of the farms involved.

4. These observations are discussed in relation to botulinus spore distribution, and to the prevention, diagnosis and treatment of the disease.

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RÉSUMÉ

Dans cet article, l'auteur rapporte 6 nouveaux cas de botulisme. Les enquêtes menées dans chaque cas démontrèrent que l'agent étiologique provenait de venaison, de blé d'Inde et d'épinards en conserve. A date, 63 personnes au Canada ont déjà été atteintes de botulisme; 35 d'entre elles sont mortes, donnant un taux de mortalité de 55.5%. La majorité des cas se sont présentés dans l'Ouest du pays. Les quatre principales mesures préventives comportent: le lavage et le triage des fruits et des légumes destinés à la mise en conserve à la maison; le choix de récipients appropriés (de préférence, en verre, et à couvercle vissé); la cuisson à 250° F. pour 70 minutes au moins, avant la mise en conserve; et enfin, le réchauffage à l'ébullition pour 10 à 15 minutes, immédiatement avant de servir les aliments. Le diagnostic du botulisme aigu ne devrait dérouter aucun praticien bien informé. Les cas sous-aigus, surtout s'ils sont isolés, peuvent cependant présenter quelques difficultés. Le taux de mortalité très élevé au Canada pourrait être réduit à des proportions comparables à celles que l'on trouve en Europe, si le diagnostic était facilité par la mise en garde du médecin vis-à-vis d'une pareille éventualité. Le traitement est basé sur l'emploi d'antitoxine et de toxoïde. Aucune maison canadienne ou américaine ne produit actuellement d'antitoxine polyvalente. Le traitement symptomatique, en particulier, de l'état de choc des intoxications aigües, occupe une place importante dans la thérapie. M.R.D.

LABORATORY STUDIES ON DISINFECTION OF ORAL THERMOMETERS

Test thermometers were contaminated with a thin layer of sputum and then placed in an incubator for thirty minutes. Following a partial drying, the thermometers were either wiped with dry cotton, wiped with different soap solutions or not wiped. After rinsing, the thermometers were placed in varying concentrations of ethyl alcohol, isopropyl alcohol, formalin, alcoholic iodine solutions, aqueous iodine solutions or solutions of quaternaries. The results indicated that the most effective cleaning procedure was thorough wiping with clean cotton wet with a solution of equal parts of 95% ethyl alcohol and tincture of green soap. Following thorough cleaning, immersion of the contaminated thermometer in 0.5 to 1.0% solution of iodine in 70% ethyl alcohol or in 70% isopropyl alcohol for 10 minutes will reduce the probability of viable bacterial pathogens of the respiratory tract remaining on the thermometer to a very low level. Aqueous iodine solutions, 70% ethyl alcohol and 90% isopropyl alcohol were nearly as effective when preceded by an effective cleaning procedure.—L. Sommermeyer and M. Frobisher, Jr.: *Nursing Research*, 1: 32, 1952.

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TWO CASES OF SPONTANEOUS RUPTURE OF ŒSOPHAGUS

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SPONTANEOUS RUPTURE of the œsophagus can be defined as sudden rupture, not associated with direct trauma, of a healthy œsophagus or an œsophagus weakened by disease. Some authors include rupture due to indirect trauma; this is not strictly accurate, but since both these types of rupture present identical clinical pictures and require the same treatment they can be considered together. Other authors have excluded those cases of rupture wherein the œsophagus had apparently been weakened by preceding disease, usually œsophagitis; the presence or absence of preceding œsophagitis cannot always be established, so the exclusion of those cases related to previous œsophagitis is not warranted. Spontaneous rupture is usually precipitated by vomiting or attempting to vomit.

CASE 1*

A 28 year old farmer was thrown to the ground when his tractor overturned on June 29, 1953, apparently receiving a blow to his abdomen. Immediately thereafter he developed abdominal pain which became increasingly severe. Examination on admission to Wilkie Union Hospital revealed exquisite tenderness across the upper abdomen, most intense in the upper left quadrant, with associated board-like rigidity. He had great pain on breathing, with marked dyspnœa and a dusky cyanosis confined mainly to the face. The blood pressure was well maintained at 128/78. No free air was demonstrated in the abdomen in lateral decubitus film. There was no evidence of surgical emphysema. He was operated upon (by F.T.P.) four hours after admission with a provisional diagnosis of ruptured spleen; complete exploration of the abdomen revealed no abnormalities. His condition remained relatively unchanged until the following day when he suddenly died at 3.30 p.m. Autopsy showed 50 c.c. of dirty brown fluid in the left pleural cavity, considerable pleuritis and a 2 cm. rent in the lowermost end of the left side of the œsophagus.

The first case then is a case of ruptured lower end of the œsophagus due to indirect trauma and characterized by severe abdominal pain and rigidity, marked dyspnœa, cyanosis, but no surgical emphysema. The diagnosis was established at autopsy.

*The first case was treated by F.T.P. and D.J.B. at Wilkie Union Hospital. Only four weeks later D.J.B. was called to see the second case and immediately diagnosed rupture of the œsophagus. This case was referred to Saskatoon City Hospital for treatment.

CASE 2

Miss L., 55 years old, was admitted to Wilkie Union Hospital at 3.20 a.m., July 25, 1953. After supper on the previous night she had experienced minor nausea, "like I was getting stomach flu." Over the past several years she had suffered several minor attacks of epigastric distress relieved by taking baking soda. After retiring early she awoke at 2.00 a.m., very nauseated; she attempted to vomit, in vain, and immediately developed severe pain in the lower substernal area with radiation through the back in the interscapular area. Examination revealed marked tenderness in the upper abdomen with board-like rigidity, severe dyspnœa, and marked cyanosis; there was a well marked cervical surgical emphysema most obvious on the left side and extending from the sternal notch to near the zygoma on the left side; the pulse rate was 100 and blood pressure 112/68. The cyanosis was most marked over the face and did not yield to oxygen by nasal catheter. Chest examination, initially negative, later revealed signs of fluid accumulation in the left pleural cavity.

Tentative diagnosis of spontaneous perforation of the œsophagus was made and the patient was transferred to the Saskatoon City Hospital. After this trip of 130 miles she was diffusely cyanosed, very severely dyspnœic with a pulse rate of 120-130, and blood pressure maintained at 130/80. Chest radiograph showed a massive left hydro-pneumothorax. Aspiration of the chest revealed a tension pneumothorax, and the presence of what appeared to be gastric juice. Preoperative transfusion with 1,000 c.c. of plasma and administration of oxygen by nasal catheter produced no improvement in the clinical condition.

Left thoracotomy was performed eight hours after onset of symptoms; a three cm. linear tear with a 1.5 cm. transverse component in its lower part was discovered in the lowermost portion of the left side of the œsophagus. This was sutured in two layers with interrupted 4-0 black silk. The mediastinal pleura was opened and the mediastinal and pleural cavities were thoroughly cleansed of all free fluid and food.

The immediate postoperative condition was excellent; the cyanosis and marked dyspnœa disappeared and the patient's general condition was greatly improved. Gastric tube suction was utilized for two days, followed by gastric tube feeding until the sixth day. Two days after removal of the tube an œsophageo-pleuro-cutaneous fistula developed which closed spontaneously in six weeks. Associated with this was a small empyema cavity which gradually disappeared following drainage. Her nutrition was maintained during this period through a small intranasal polyethylene tube passed into the jejunum after the technique of Fallis and Barron.[15] Antibiotics were used generously during the early postoperative weeks. She was discharged on Oct. 2 in good health and has remained well since. Barium swallow examination six weeks later showed a completely normal œsophagus. Gastric radiographs showed no evidence of peptic ulcer, although this patient's previous history was suggestive of duodenal ulcer.

This second case is one of spontaneous rupture of the lower end of the left side of the œsophagus treated by thoracotomy and repair eight hours after onset of symptoms. An œsophageo-pleuro-cutaneous fistula developed but the patient went on to complete recovery. It is felt, in view of the reputation the œsophagus has for slow healing, that if tube feedings had been continued for a total of 10 days the fistula might not have developed. Anderson[3] and Ware[47] have suggested that tube feedings be continued for a total of 10 days.

DISCUSSION

Anderson, Mackler, and Ware have excellent reviews[3, 27, 47] on spontaneous rupture of the œsophagus. Anderson records 108 cases with survival in 30. Since Barrett[4] reported the first survival following thoracotomy in 1947 an increasing number of cases have been reported, and, since Anderson's review, there are an additional 27 reported cases[6-8, 17, 21, 22, 24, 25, 27, 29, 31, 34, 40, 43-47] and 14 survivals, making a total, including our case, of 136 cases with 45 survivals.

The recent increase in the number of cases reported suggests that this condition is not so rare as might be suspected, and that perhaps in the past it has often gone unrecognized. The diagnosis may be missed even at autopsy, the cause of death being considered to be empyema: the reason for this is that the stomach may be sectioned from the œsophagus exactly at the site of perforation, so that the perforation is unrecognized.

Sudden ruptures, apart from direct trauma, occur either with indirect trauma, as in case 1, or "spontaneously" after vomiting, as in case 2. Spontaneous rupture of the œsophagus has occurred other than post-emetic, i.e. after drinking a glass of water,[28] during defæcation,[35] or during convulsions.[23]

REVIEW OF LITERATURE ON SPONTANEOUS PERFORATION OF THE ŒSOPHAGUS

We have reviewed 53 recent cases.[3-8, 11, 12, 17, 18, 21, 22, 24-27, 31, 33, 34, 37-40, 42-45, 47] Vomiting or attempting to vomit was the initiating factor in practically all cases. Twenty-three or approximately 44% gave a history of preceding peptic ulcer or one suggestive of peptic ulcer. In only 12 cases was there a history of preceding alcoholic indulgence, and amongst these not all gave a history of recent alcoholic indulgence. Overeating was not a major factor in these cases. Ware[47] also minimizes the importance of overeating and alcohol.

Forty-seven of the 53 patients were over 40 years of age. It is perhaps significant that no patient over 70 years of age survived. Forty-two of these cases, or approximately 80%, occurred in males. Eliason and Welty[14] in an earlier review reported 86% of cases in males.

With regard to the site, 48 of the 53 cases were supra-diaphragmatic and 41 of these were on the left side; the remaining seven cases were on the right side or were anterior or posterior. There were five cases at other levels than strictly supradiaphragmatic: three were in the middle third of the œsophagus, one was in the lower third, and one was in the cervical œsophagus—the latter[40] appears to be the only such case reported. Cervical surgical emphysema was present in 28 or approximately 50% of cases. Twenty-seven of these patients survived, so that the current survival rate in reported cases is almost exactly 50%.

ETIOLOGY

As mentioned previously there is no doubt that many of these cases are associated with œsophagitis: others apparently are not. Three factors are common in the past history of these patients (1) Indigestion of a peptic ulcer type or definite ulcer history. (2) Alcoholism. (3) Overeating. The significance of these is discussed above. They can be instrumental as etiological factors, either by being associated with œsophagitis or by inducing vomiting.

The predominance of rupture at the lower end of the œsophagus is because this is the weakest portion of the viscus. McKenzie in 1848, and Kinsella[22] and Mackler[27] more recently have demonstrated in cadavers that the lower end is the weakest portion of the œsophagus; it will withstand 5¾ to 11 pounds pressure per square inch in adults and approximately twice this pressure in children, which no doubt partially accounts for the low incidence of rupture in children.

The high incidence of left-sided rupture at the lower end is probably related to the fact that this is the most unprotected portion; also, the œsophagus deviates to the left at this point and vomitus tends to impinge more directly on the left side of the collapsed œsophagus. In some cases vigorous descent of the diaphragm in vomiting would appear to have produced complete avulsion of the œsophagus from the stomach as in Boerhaave's case, the first case described, and as in a similar case more recently described by Higginson and Clagett.[20]

There is a separate group of spontaneous ruptures in which a neurogenic factor seems important. Such cases have been described after neurosurgery, especially involving the middle or posterior cranial fossa;[9, 16, 38] one case has been described associated with acute polioencephalomyelitis.[22] These cases, in which rupture usually occurs in the middle or lower third of the œso-

phagus[1, 22, 38] are characterized by diffuse disease of the œsophagus. Abbott[1] states that primary repair is not possible in these cases and recommends insertion of a T-tube into the œsophagus. The diagnosis should be suspected after craniotomy[16] when patients develop vomiting, hæmatemesis, and respiratory distress.

SYMPTOMS AND SIGNS OF SPONTANEOUS RUPTURE

Characteristically the patient during or after vomiting develops severe retrosternal or upper abdominal pain radiating to the back in the interscapular area; there is rapidly increasing dyspnœa and cyanosis with elevation of pulse rate; the blood pressure is usually maintained until near the end when sudden circulatory collapse develops. If the patient develops surgical emphysema in the neck—and 50% of patients do—the diagnosis is almost certain. Chest radiography, in the absence of rupture into the pleural space, will show mediastinal emphysema, or widening of the mediastinum, or increase in the normal spacing between œsophagus and spine, which is usually no wider than the tracheal column of air.[13] With rupture into the pleural cavity a hydropneumothorax develops, which in some cases is bilateral. Lipiodol swallow may be used to confirm the diagnosis. Aspiration of the chest may show the presence of gastric juice, and also will relieve any tension pneumothorax that may be present.

DIFFERENTIAL DIAGNOSIS IN SPONTANEOUS RUPTURE

The differential diagnosis includes: (1) Spontaneous mediastinal emphysema. (2) Ruptured bronchus with surgical emphysema. (3) Perforated peptic ulcer. (4) Acute pancreatitis. (5) Spontaneous pneumothorax. (6) Spontaneous hæmopneumothorax. (7) Perforated stomach in a diaphragmatic hernia. (8) Pneumonia or empyema, if the case is seen in the late stages.

Perforation of the stomach in diaphragmatic hernia is treated similarly and would be indistinguishable except at operation. The most common error in diagnosis is to consider these cases as cases of perforated peptic ulcer. Spontaneous hæmopneumothorax, also a rare condition, is perhaps the most difficult condition to exclude in the differential diagnosis, especially if surgical emphysema is absent.[30] However, in spontaneous hæmopneumothorax vomiting does not generally initiate symptoms, the abdominal signs are little in evidence, and Lipiodol swallow or aspiration of the chest cavity will decide the diagnosis if ruptured œsophagus is present.

Rupture of the œsophagus from indirect trauma.—The only difference in the clinical history and progress of a case due to indirect trauma, as in the first case reported here, is the history of trauma, after which the patients develop the signs described above. Mackler has reported one case due to indirect trauma and reviewed three other cases,[2, 32, 36] finding in all of these cases except one that the rupture was in the lower end of the œsophagus on the left side.

TREATMENT

As regards treatment, these cases of sudden perforation of the œsophagus may be divided into two groups: (1) Those seen early, within the first 12 to 24 hours. (2) Those seen later, who have weathered the initial period of shock and have a large hydropneumothorax or mediastinal loculation.

Most of the cases seen in the *first group* will die if untreated. It appears that they die from circulatory collapse due to the extreme irritation set up by the presence of gastric juice widely spread throughout the mediastinal and pleural cavities. Many authors[27, 41, 48] attribute the improved clinical condition after surgery to the thorough cleansing of the mediastinal and pleural cavities, so that there is no longer pleural or mediastinal irritation or compression from gastric juice or air. Certainly in our second case this would seem to have been true.

As soon as the diagnosis has been made gastric suction should be established. The accepted treatment in the first group is by thoracotomy and repair of the rupture, using two layers of interrupted 4-0 black silk. Lipiodol swallow may be used to determine the side or site of rupture. The mediastinal pleura should be widely opened with cleansing of the mediastinal and pleural cavities, including removal of all food particles and irrigation with normal saline. Postoperatively these patients receive routine thoracotomy care: in addition patients should be allowed nothing by mouth, nutrition being maintained by gastric tube feeding.

Several authors have reported fistula formation after repair. These fistulæ have not created any great problem, and as a rule heal spontaneously with local thoracic drainage and œsophageal

rest by tube feeding. Maintenance of full re-expansion of the lung is essential in prevention of empyema. Whereas it is true that the recovery of these cases treated by primary suture may be marred by complications such as fistula or empyema, there is an increasing number of cases in which uneventful recovery is obtained and the patient is discharged within two weeks.[7, 26, 29, 41] Further experience with these cases should increase the incidence of uncomplicated primary healing.

The *second form of treatment* is applicable to cases that are not seen or diagnosed in the first few hours of the disease: a provisional time limit of 24 hours might be set. A certain number of these patients will survive the initial stage of shock, and will be seen later with a large hydropneumothorax, an empyema, or a mediastinal abscess. These cases can be treated by thoracotomy with cleansing as best possible, and drainage of the pleural cavity and mediastinum with re-expansion of the lung. The patient should then be fed parenterally or by jejunal or gastric tube. Gastric feeds given by tube or by gastrostomy may be regurgitated through the œsophageal opening.[19] It is then necessary to institute jejunal feeding, which can be done without jejunostomy by the Fallis and Barron technique.[15] The patients also require, of course, vigorous antibiotic therapy. They will no doubt develop an œsophageo-cutaneous fistula through their empyema cavity, which, with continued tube feedings and adequate empyema drainage, will gradually close over in a matter of weeks or even sooner. In some cases[3] late decortication and late repair of the rupture have been performed. Delayed repairs, however, have almost invariably broken down, and, as experience has shown that these fistulæ heal with time, it would seem wiser not to attempt delayed repair or secondary repair.

SUMMARY

Two cases of ruptured œsophagus are reported; one fatal, due to indirect trauma; the second of spontaneous origin, with recovery. Fifty-three recent cases in the literature are reviewed.

Both these types of rupture present similar clinical findings with sudden chest and abdominal pain, severe dyspnœa, cyanosis and abdominal rigidity. Examination will reveal cervical surgical emphysema in about 50% of cases, and hydropneumothorax in most cases. Lipiodol swallow may be used to confirm the diagnosis and locate the site of rupture, which is usually a linear tear on the left side of the lowermost part of the œsophagus.

The treatment of choice is immediate thoracotomy and repair of the perforation. Cases diagnosed late (after 12 to 24 hours) are probably best treated with thoracotomy and cleansing of the pleural and mediastinal cavities as well as possible, with drainage and as a rule without repair of the rupture, as repair at this stage is almost invariably unsuccessful.

Œsophago-pleuro-cutaneous fistulæ are common complications; these will close spontaneously.

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ISONICOTINIC ACID HYDRAZIDE IN THE TREATMENT OF TUBERCULOUS TENOSYNOVITIS*

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TUBERCULOUS TENOSYNOVITIS may be defined as a specific chronic infection of the tendon sheaths of the hand or foot. It is a relatively rare form of tuberculosis. At the Jewish General Hospital, there has been only one proven case in over 90,000 admissions. At the Mayo Clinic, Bickel[1] and his co-workers reported 37 proven cases in a survey extending over 34 years. Adams[2] and his group at the Massachusetts General Hospital reported a virtually similar incidence: 30 cases in 45 years.

Treatment up to the present time has consisted of two main types used alone or in conjunction: (1) Conservative: i.e., the general treatment of tuberculosis: rest, immobilization, good food, heliotherapy, and within the last few years, the administration of specific antituberculosis medication. (2) Radical: excision of the diseased tissue.

The latter is quite a formidable procedure, however, often attended by untoward results, such as damage to tendons and nerves. Even with operation, the course of the illness is generally prolonged, splinting for months being necessary, with resultant stiffening of adjacent joints. Moreover, there is the constantly present danger of a flare-up, particularly if some of the diseased tissue has been overlooked at the time of the original operation.

Bunnell[3] reports 21 cases, all treated by operation. In this series, 14 patients were cured and six had local recurrences; of the latter, two died from generalized tuberculosis. One case could not be traced. At the Mayo Clinic,[1] the only treatment found to be of definite value was surgical excision of the lesion. Steindler[4] recommends conservative treatment in children, but cautions that although it often yields good results, it is long and tedious. For adults, he advocates complete extirpation of the tendon sheath. Iselin and Vassitch[5] regard operation as the treatment of choice, and employ streptomycin, PAS, and isonicotinic acid hydrazide preoperatively to induce localization, and postoperatively to prevent recurrences. Miller *et al.*[6] report two cases of tuberculous tenosynovitis treated successfully with streptomycin alone, without immobilization or operative intervention.

It was thought to be of general interest to report in detail a case of proven tuberculous tenosynovitis in an adult treated conservatively, in which arrest of the active process and apparent cure could be attributed to the use of isonicotinic acid hydrazide.

W.F., a white man, aged 45 years, admitted to the Orthopædic Service of the Jewish General Hospital on January 26, 1952, noted several weeks before the development of a swelling on the volar surface of the left forearm. There was no history of antecedent trauma. The mass seemed to be increasing in size slowly and was not particularly painful. However, its indolent nature soon alarmed him, and he applied for admission.

He had been under treatment at the Jewish General Hospital in 1948 for tuberculosis of the fourth and fifth lumbar vertebræ. A bone graft fusion was performed and he had remained well and had been able to work continuously as an elevator operator.

On admission, the distal part of the volar surface of the left forearm presented an oval swelling about two and one-half inches in its wide diameter. Two small draining sinuses were present in the centre of this swelling. These sinuses had apparently followed the too vigorous application of a liniment locally. Distal to the swelling and continuous with it, there was considerable induration of the mid-palmar space and hypothenar eminence. The fingers were flexed, and movements restricted and painful with the exception of the thumb. The general physical examination was essentially negative. The Mantoux 1:1000 reaction was strongly positive. There was a moderate shift to the left of the neutrophils and a slight lymphocytopenia. The Kline test was negative. The smear from the sinuses showed Gram-positive rods and Gram-negative cocci. Culture yielded *Bact. coli* and hæmolytic *Staph. aureus*. X-ray examination of the forearm and hand showed considerable soft tissue swelling but no bone involvement. Radiographs of the lumbar spine revealed little change from the one taken in 1948. The lesion appeared healed and the bone graft intact.

The findings of an indolent swelling involving the tendon sheaths of the hand with secondary infection, with the antecedent history of tuberculosis of the lumbar spine, suggested a tentative diagnosis of tuberculous tenosynovitis.

Therapy during his first admission consisted of administration of penicillin, 1,500,000 units, streptomycin, 7,000,000 units, para-aminosalicylic acid, 21 gm. and chloramphenicol, 3,000 mgm. Locally, hot saline fomentations, bacitracin dressings and ultraviolet rays were employed.

However, there was no real improvement in the patient's condition. On February 2, one week after admission, we were obliged to discharge him to the out-patient department on account of family obligations. There his forearm and hand were immobilized in a plaster of paris cast, and penicillin 600,000 units and streptomycin 1,000,000 units were administered daily. This treatment was continued until February 27, 25 days later, when the cast was removed because of severe pain. It was then found that the entire hand was badly swollen, that there were no active movements in the fingers, and that the swelling on the volar aspect of the forearm had broken down, leaving an ulcerated area about two inches in diameter.

Tissue from the ulcer was sent for pathological examination, and was reported on as follows: "Specimen consists of a number of small irregular fragments of tissue which are cartilaginous in consistency, transparent, and of whitish grey colour. Microscopic section reveals a

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number of irregularly shaped masses of amorphous, polychromatic and acellular material, along the periphery of which small numbers of polymorphonuclear leukocytes are enmeshed in amorphous material. No fixed structures of any type can be identified. No granulation tissue or giant cells are present and no caseation necrosis is seen."

The patient was again admitted to the hospital on March 1, and placed on antibiotic therapy consisting of penicillin and streptomycin by injection and bacitracin locally. Four days after admission, the sensitivity tests showed a definite response to terramycin and chloramphenicol. Accordingly, he was placed on chloramphenicol 1,000 mgm. daily until his discharge 22 days later, again for family reasons.

During this second admission the question of surgical excision of the lesion was considered, but, as the process appeared active and progressive, with definite signs of toxicity such as continued elevation of temperature, it was decided to await localization.

The patient was again referred to the out-door department where he was treated in the same manner as during his second admission. There, another culture was taken from the ulcer on the forearm and, for the first time, was reported positive for *Mycobacterium tuberculosis*.

Two weeks later, on April 15, the patient's condition again necessitated admission. Locally, the ulcer on the volar surface of the forearm was still discharging, there was considerable pain and marked restriction of the movements of all the fingers, swelling over the volar aspect of the wrist, and tenderness and swelling of the hypothenar eminence. Despite the bacteriological evidence for a diagnosis of tuberculous tenosynovitis, streptomycin was no longer given, owing to its proven ineffectiveness in this case. Hot compresses were applied locally and chloramphenicol 1,000 mgm. daily administered by mouth. On April 18, 10 weeks following the first admission, isonicotinic acid hydrazide became commercially available for the first time, and the patient was immediately started on 2½ tablets (250 mgm.) daily. All other treatment was discontinued (see Fig. 1).

By April 22, only four days after commencement of the use of the drug, the hand had become less œdematous, and there was definite lessening of discharge and beginning healing of the wound. The patient was then discharged from hospital with instructions to continue to take isonicotinic acid hydrazide in the same dosage daily. Examination 13 days later revealed a marked decrease in the size of the swellings over the anterior aspects of the wrist and hypothenar eminence, and virtually no discharge from the ulcer.

Twenty days after isonicotinic acid hydrazide treatment began, the ulcer in the forearm was closing and becoming covered with a crust. The swelling of the palm was noticeably smaller. There was still some weakness of the fingers, but the grip was definitely stronger. Subjectively, there was also great improvement. The patient no longer felt sick as heretofore. Every time he was seen in the clinic, he requested permission to go back to work.

On May 29, 41 days after commencing treatment, the ulcer was practically closed; the induration of the hypothenar area was almost gone; the fingers moved freely, except the little finger, in which movement was still somewhat restricted. There were no systemic symptoms such as fever or anorexia. At no time, although constantly on the watch for them, did we see any of the toxic

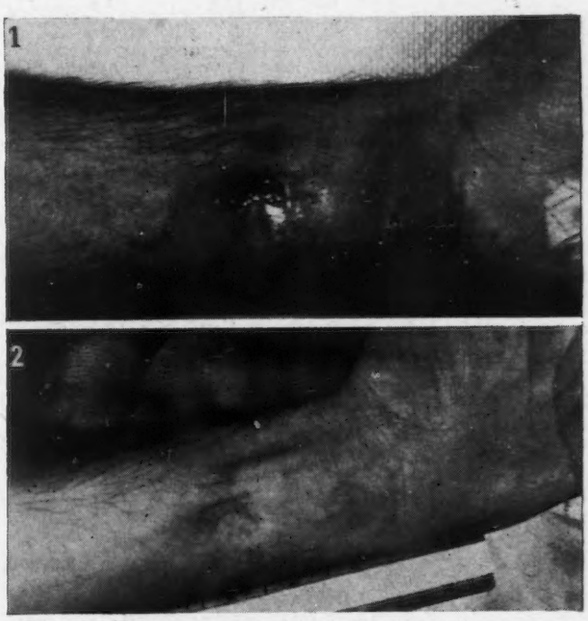

Fig. 1.—Forearm and wrist at the time of commencement of isonicotinic acid hydrazide therapy. Fig. 2.—After treatment with isonicotinic acid hydrazide.

manifestations listed by Selikoff[7] and his coworkers as complicating isonicotinic acid derivative therapy, such as vertigo, drowsiness, headache, dysuria or dryness of the mouth. Accordingly, it was thought safe to continue administration of the drug for three and a half months, during which time repeated examinations of blood and urine gave normal results. By this time, there had been a return to normal in every respect, except for a slight restriction in flexion of the little finger (see Fig. 2).

SUMMARY

A case of tuberculous tenosynovitis of the forearm and hand has been presented. While awaiting localization before attempting surgical eradication of the lesion, all types of conservative therapy were employed: penicillin, strepto-

mycin, chloramphenicol, para-aminosalicylic acid, hot saline fomentations, bacitracin ointment, ultraviolet rays and plaster of paris immobilization, without any appreciable effect. The administration of isonicotinic acid hydrazide,[8] the therapeutic properties of which were discovered in the systematic screening of the thiosemicarbazones and related substances for anti-tuberculosis activity, produced a rapid remission and apparent cure of a condition otherwise protracted if not intractable.

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RÉSUMÉ

Nous avons présenté un cas de ténosynovite tuberculeuse de l'avant bras et de la main. En attendant que les lésions se limitent pour en permettre l'excision, nous avons épuisé tout l'arsenal thérapeutique médical: la pénicilline, la streptomycine, la chloromycétine, le PAS, les compresses chaudes, l'onguent de bacitracine, les rayons ultra-violets, et l'immobilisation plâtré, sans succès. L'usage de l'hydrazide de l'acide isonicotinique dont la valeur thérapeutique s'est révélée au cours de recherches systématiques sur les effets bactériostatiques des thiosemicarbazides et des substances apparentées dans la tuberculose, a amené une régression rapide et même une guérison apparente d'une condition qui, si elle n'était pas tout à fait incurable, se prolongerait démesurément.

A STUDY OF PROTEIN REPLETION IN A CASE OF MARKED UNDERNUTRITION*

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THROUGHOUT THE WORLD severe undernutrition is usually the result of famine. In Canada it is nearly always the result of functional or organic conditions that interfere with ingestion, digestion, absorption and storage of nutrients, or it is due to diseases which alter the metabolic rate. Treatment is important in re-establishing the body economy and in preventing the diseases to which a poorly nourished individual is susceptible. However, undernutrition as an entity is often poorly managed for several reasons:

1. There are cases in which the diagnosis of undernutrition is masked by a primary disease and most of the physician's efforts are directed to recognizing the underlying condition. Should the physician ignore the nutritional status in such patients he may discover later that the primary disease is not amenable to therapy other than improving the nutritional state. It should be remembered that if the primary cause of the poor nutrition requires surgical intervention, the morbidity and mortality are proportional to the state of nutrition, other things being equal.

2. Undernutrition may at times present such a fearful clinical picture that it may be assumed to be the result of a hopeless cancer, with resultant apathy in investigation and treatment.

3. Treatment of severe undernutrition, although appearing superficially simple, may well be difficult. There is often a slow response when complicating disease is present and fulfilling the body's caloric needs in such cases is a problem that tests the ingenuity and patience of the therapist.

The case presented is one that demonstrates many of these points.

A 58 year old white male was admitted to hospital on June 23, 1952, with the following complaints: (1) Intermittent swelling of the legs—one year. (2) Intermittent nausea, vomiting and diarrhœa—one and a half years. (3) A loss of 30 pounds in two years.

Two and one-half years before admission a gastrectomy was performed for a radiologically proven gastric ulcer complicated by pyloric stenosis. Six months later he began to experience nausea immediately after meals, accompanied by a feeling of fullness. Intermittent attacks of vomiting and diarrhœa soon followed, the diarrhœa being greatly intensified by increasing fat intake and the stools being described as greasy. One year prior to admission he was investigated at the hospital where the operation was performed. No improvement occurred, although by this time he had noted dependent œdema and was told he was slightly anæmic. One month before

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admission he visited his family physician with complaints of fatigue, loss of weight and dyspnœa; the latter symptom appeared to be due to a large left pleural effusion. He was admitted to hospital after a thoracentesis had been done. His past history otherwise was non-contributory and he specifically denied having any other respiratory symptoms or exposure to tuberculosis.

On physical examination the man was described as pale and emaciated—he was lethargic but well oriented. There was marked pitting œdema of the legs. The pulse rate was 90, B.P. 110/60, temperature 99° F. There was impaired resonance of the left hemithorax with diminished and absent breath sounds. Examination otherwise was non-contributory.

Following a thoracentesis, a chest radiograph revealed infiltration and cavitation of the left upper lobe. He remained in that hospital until November 14, 1952, a matter of nearly five months and was thoroughly investigated in an unsuccessful attempt to diagnose his lung disease. Cultures of gastric washings and pleural fluid were persistently negative for tubercle bacilli and a bronchoscopy was negative. There was low grade fever every day and, because of a tentative diagnosis of tuberculosis, streptomycin and PAS were tried and abolished his fever. The gastro-intestinal symptoms continued and vomiting often occurred. He had a barium meal which demonstrated a narrow stoma with retention of the barium proximally. All such studies were performed with difficulty because he had developed a tendency to faint on slight provocation. The œdema did not improve although there were temporary regressions with the use of mercurial diuretics. The total serum protein value towards the end of this admission was reported as four gm. %, and the albumin level was said to be low but not specifically reported. His condition just before his transfer to Shaughnessy Hospital was critical. This may be gathered from the fact that a metacarpal fracture he incurred in fainting was not reduced because he was considered such a poor anæsthetic risk.

He was transferred to the chest unit of Shaughnessy Hospital on November 14. He now had anasarca and ascites, his skin was pigmented and there were areas of ecchymosis. The tongue was red and smooth and signs of a left pleural effusion persisted. His Hb value was 63.5% with slight macrocytosis, the total serum protein value was 3.65 gm. %, albumin was 0.85 gm. %. The electrolyte levels were within normal range with the exception of calcium which was 7.6 mgm. %. Urinalysis and liver flocculation studies gave negative results. Total fat in the stools was more than 65% of the fat intake. Prothrombin time was 35% of normal. Barium meal again revealed delay of food entering the stoma and also segmentation of barium in the small intestine. He received vitamin B complex and vitamins B_{12} and K parenterally. Streptomycin and PAS were administered although a positive diagnosis had not yet been made. Efforts were made to improve his intake and he was placed on six feedings daily of a diet that was high in protein and contained 2,500 calories. His condition continued to deteriorate, he became even more œdematous, and day in and day out he continued to vomit.

On January 21, 1953, he was transferred to the Clinical Investigation Unit of the hospital because of his failure to respond. On January 23, a Miller-Abbott tube was passed but it did not enter the stoma and was removed the next day. He continued to vomit and at this time received his third blood transfusion since admission.

On January 30, nitrogen, fluid, sodium and potassium balance studies were commenced. The nitrogen was determined by the macro Kjeldahl method, the electrolytes by the flame photometer and the technique used was that of Reifenstein, Albright and Wells.[1] The intake and output of these substances were observed in this manner for 119 days (see Fig. 1).

On February 2, a weighted polyvinyl tube was passed into the jejunum and he was fed by continuous drip for 67 days. The jejunostomy feedings at first contained only 1,100 to 1,300 calories because of gastro-intestinal symptoms. Within two weeks it was possible to increase this to 1,600 and 1,700 calories daily. Supplementary oral feedings were added after three days so that the total caloric intake increased from 2,000 at the beginning to 3,200 during the latter days of the study and the protein content of the diet increased from 120 gm. to 180 gm. daily. Vitamins administered daily through the tube included 30 mgm. thiamine, 125 mgm. niacinamide, 10 mgm. riboflavin, 300 mgm. vitamin C and 75 mgm. of vitamin K.

After the first few days of the jejunostomy feedings he ceased vomiting. At the beginning of the third week he had a marked diuresis which persisted for three to four weeks during which he lost 12 kg. of œdema fluid (Fig. 1). His condition improved strikingly over the following weeks. He became keener, stronger and happier. He remained in positive nitrogen balance throughout. His Hb at the 20th day of balance was 54% but at the end of the study was 85%. No iron therapy was given. The serum albumin value was 1 gram % at

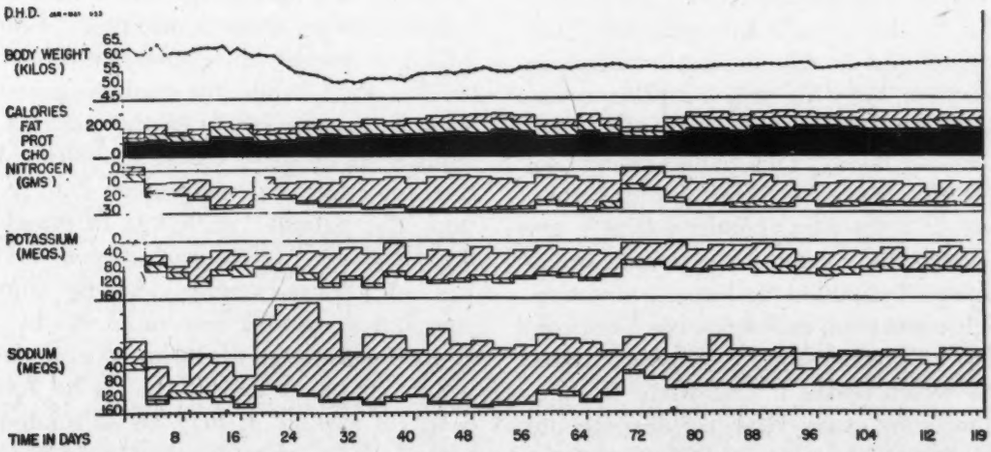

Fig. 1.—In regard to the nitrogen, potassium and sodium balance charts, the intake is represented between the base line at zero and the lowest horizontal line below the base line, whereas the output may be calculated from the lowest horizontal line to the uppermost horizontal line. When the latter is above the base line the subject is in negative balance and when below is in positive balance. The various output partitions are explained by the following legend.

Caloric intake legend.
\ — Fæcal excretion.
/ — Urinary excretion.
■ — CHO (carbohydrate).
\ — Protein.
/ — Fat.

the beginning of the balance study; by the third week, when the diuresis began, it had increased to 1.8 gm. %. At the end of the study the total serum protein value was 6.9 gm. % and the albumin 3.3 gm. % (See Fig. 2).

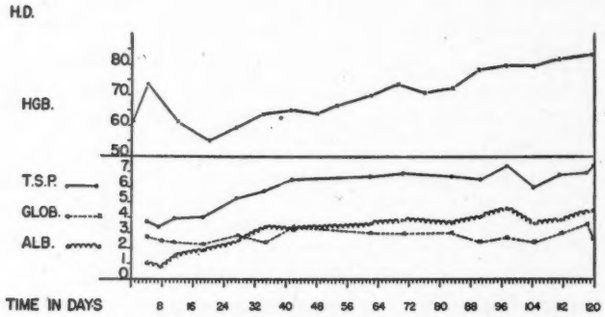

Fig. 2.—Serial hæmoglobin and blood protein studies.

During the early days of the balance study he produced sputum which on the twelfth day was found to contain tubercle bacilli. He was then placed on streptomycin and PAS.

His weight at the beginning of the study was 62.5 kg.; he was at this time grossly œdematous. At the end of the balance study it was 57.5 kg. and the œdema had disappeared.

After the tube was removed there were no gastro-intestinal symptoms. He is now a patient in the chest unit where his tuberculosis is responding to chemotherapy and rest. At the present time, some five months after these studies have been completed, although approximately 10 pounds lighter than before his operation, he looks well and has no symptoms. The percentage of ingested fat excreted in the stool is now normal, being less than 10%.

DISCUSSION

This case therefore, was one of severe undernutrition complicated by pulmonary tuberculosis. There is no positive evidence that the latter condition did not exist before gastrectomy. Clinically the tuberculosis appeared to be a complication of his nutritional state. Pulmonary tuberculosis is notorious[2] for attacking the undernourished and in this patient it found an excellent medium. Why his gastric washings were not positive is conjectural. Sputum began to appear in the early days of his improvement. He stated that he was too weak to bring it up before.

The basis of this man's undernutrition appeared at first to be a poorly functioning stoma with resultant obstruction and semi-starvation. But since his nutritional deficiency has been corrected, the gastro-intestinal symptoms have disappeared which makes it impossible to diagnose the antecedent cause. However, it was quite clearly a result of his operation and due to gastro-intestinal dysfunction.

Œdema in undernutrition is often evident in the absence of a signficant fall in the serum albumin level. The cause of this type of œdema has never been established. McCance[3] believes that one of the most important factors is the simple replacement by fluid of the spaces left by atrophy of fat and other tissue cells, causing a relative increase in extracellular fluid, large enough to produce dependent œdema.

Many other factors have been considered to play a part but they are either highly theoretical or are backed by flimsy evidence. In this case, the major cause was the very low level of albumin which was far below the so-called "critical level" of colloid osmotic pressure; therefore, as the albumin level rose, the œdema disappeared. In experiments on dogs[4] subsisting on protein deficient diets, the amount of nitrogen retained to increase the serum albumin value by 1 gm. % was calculated. From these experiments it has been estimated that in man 750 gm. of protein is retained to increase the serum albumin value 1 gm. %. In this study, when the serum albumin level was low, the amount of protein retained during a 1 gm. % increase ranged from 650 to 1,030 gm. This correlation demonstrates the large amount of protein necessary to be retained by the depleted body before the albumin level rises significantly, and indicates the many weeks of treatment required before total serum protein values reach normal.

The serum sodium, chloride and potassium levels were always normal. The calcium level, however, was low; this has been the case both in studies during famine and in controlled starvation experiments. The decrease in calcium is a result of its intimate association with the serum proteins, and thus it fluctuates with them. The balance studies reveal a negative sodium balance which is merely an expression of the loss of œdema fluid, while the positive potassium balance is in proportion to the positive nitrogen balance in terms of tissue anabolism. From the time the study was undertaken until its termination, the patient lost 5 kg. in weight, which represents the difference between œdema fluid loss and tissue weight gain. By dividing the cumulative sodium loss in m.eq. by 152, the approximate value of 18 kg. is obtained for the weight of the œdema fluid. As he actually lost 5 kg. of weight, it may be calculated that he gained 13 kg. of tissue. A starved person, however, is reported to gain tissue to the extent of grams of nitrogen retained $\times$ 6.25.[5] He retained approximately 900 gm. of nitrogen and the product of this formula is 5.6 kg. The lack of correlation by these methods, therefore, does not

allow us to state the weight of tissue gained or the amount of œdema fluid lost.

The anæmia was another manifestation of his deprived state and therefore responded to diet alone, without the use of iron. These anæmias are usually normocytic or macrocytic and are almost constantly found in those who have been severely undernourished over a long period of time.[6]

CONCLUSIONS

1. The treatment of undernutrition is indispensable for the restoration of health, no matter what is the cause. Such a condition may be treated successfully only by satisfying the body's metabolic requirements. If these are not fulfilled, the patient becomes unfit to combat the underlying condition and his lowered resistance predisposes him to other diseases.

2. In undernutrition a person who has severe protein depletion will probably require to retain between 650 and 1,030 gm. of protein to produce an elevation of serum albumin level of 1 gm. %.

3. Anæmia caused by undernutrition is corrected by diet alone.

4. The original cause of undernutrition in the case presented remains obscure because, following the correction of the undernutrition, the gastro-intestinal symptoms disappeared.

SUMMARY

A case of severe undernutrition is presented in which metabolic balance studies were made over a period of 119 days. The case demonstrates how undernutrition may result from underlying conditions and may predispose the patient to further complications. The metabolic studies included investigation of nitrogen, potassium, sodium and fluid balance. These are briefly discussed with special emphasis on protein repletion.

The technical assistance of the dietitian, Miss M. Bone, and of Miss M. E. Saunders, Mr. J. McCullough and Miss A. M. Dahlke of the laboratory staff is most gratefully acknowledged.

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THE RELATIONSHIP BETWEEN FIBROCYSTIC DISEASE AND CARCINOMA OF THE BREAST*

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THE RELATIONSHIP of benign to malignant lesions of the breast is still one of the problems of present day surgery. For the purposes of this report fibrocystic disease and carcinoma will be under discussion as they are by far the commonest benign and malignant lesions of the breast.

Fibrocystic disease is known by a multitude of other terms, all of which describe a condition found in women with its highest incidence during the reproductive period. The pathological picture is basically a condition of hyperplasia involving epithelium and connective tissue of the breast, often with the growth of cysts, sometimes progressing into a condition where marked atypical epithelial hyperplasia occurs in the ducts. When this occurs it is termed, for convenience, cystic disease with benign neoplasia.

Many years ago workers such as Cooper (1831),[1] Brodie (1846),[2] and Reclus (1883),[3] distinguished between these groups of pathological changes in the breast. Early in the history of the problem there appeared a division of thought, one group considering fibrocystic disease to be a precursor of carcinoma and the other that there was no relationship to malignancy. Among the better known authorities who held the first view were Schimmelbusch (1892),[4] Cheatle (1930),[5] Cutler (1932),[6] and Warren (1940).[7] Opposing this view were an equally prominent group among which were Bloodgood (1921),[8] and 1929[9]), Hart,[10] Johnson,[11] and Campbell[12] who thought there was no relationship. Campbell[12] stated that the conclusions of the investigators were practically predetermined by the methods they used. Those who obtain their data from

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histological methods of research conclude that fibrocystic disease is precancerous while most of those who use clinical evaluation and prolonged follow-up of individual cases find that the disease is not precancerous.

The tumour clinic of the Montreal General Hospital has as one of its functions the follow-up of all patients treated for malignant disease in the hospital, as well as those with benign conditions suspected of developing into cancer. Fibrocystic disease of the breast is considered one of the latter entities.

These patients are followed up at regular intervals by recalling them to a weekly clinic or by correspondence if they are out of town. The private patients are followed by letters written to their doctor. The follow-up of fibrocystic disease has been underway for some years and is continuing. An interim report was thought to be of interest because of the controversy already referred to.

METHODS

The clinical approach was used in this study; the records of the tumour clinic and pathological department of the Montreal General Hospital were consulted to establish the following: (a) The incidence of previous operations for fibrocystic disease in a series of 313 cases of proven carcinoma of the breast. (b) The incidence of previous operations for fibrocystic disease in a control series of 300 women of approximately the same age distribution. (c) The number of carcinomas which developed in a series of cases of known fibrocystic disease which had been followed up from eight to 23 years.

(a) A series of 313 cases of carcinoma of the breast in females was obtained from the records of the Montreal General Hospital tumour clinic and the pathological records. These had all been treated by some form of surgery and some by preoperative and many by postoperative irradiation therapy. In this group the incidence of fibrocystic disease found at pathological examination was 26%. Their individual case records were carefully searched for any history of previous operations on either breast for non-suppurative, non-cancerous lesions of the breast. Seven such patients were found who had previously been operated upon for non-suppurative, non-cancerous lesions of the breast. These were divided into three with a previous operation on the breast in which cancer later developed and three others who had the operation on the opposite breast. One of these seven had had two previous operations, one on each side. Thus there were four patients in all with a previous operation on the same breast (1.27%) and four with the operation on the opposite breast.

Since fibrocystic disease is usually considered to be a bilateral condition,[13] the seven cases may be used, giving an incidence of 2.23% of the 313 cancer patients. This percentage may be compared with other published series by Foote and Stewart[14] who found 12 patients (4%) out of 300 cancer patients in one group and 29 patients (2.4%) out of 1,200 cancer patients in another group. Johnson[11] reported two (0.45%) out of 444 cancer patients and Campbell[12] four (4.54%) out of 88 cancer patients.

(b) A control series was collected by examining the records of 300 women admitted to the Montreal General Hospital during the year 1951. These women, of age distribution similar to the 313 cancer patients, were in hospital for conditions other than malignancy or disease of the breast. Nine of the 300 had a history of a previous admission to a hospital for operation for a non-cancerous, non-suppurative lesion of the breast. Thus there were nine or 3% of the control series of 300.

A similar study has been made by Foote and Stewart,[14] who found 13 out of 1,200 women with previous operations on the breast. This incidence was 1.08%.

If fibrocystic disease is a precursor of carcinoma of the breast, one would expect many carcinomas to develop from fibrocystic breasts. This was not so in our study; there were more patients with fibrocystic disease marked enough to require operation in the control series of 300 women, than in the 313 patients with proven carcinoma.

It should be mentioned at this point that Foote and Stewart did not arrive at the same conclusion. In their series of 1,200 patients with breast cancer, 2.4% had had operations previously for fibrocystic disease as compared to 1.08% having operations for the same reason in their control series of 1,200 patients.

(c) The follow-up of 64 women with fibrocystic disease covered a period ranging from eight to 23 years, the average being 13.15 years. Their age distribution in decades was as follows: 2nd decade 1, 3rd decade 3, 4th decade 14, 5th decade 32, 6th decade 13, 7th decade 1. All case

reports were examined, the pathological diagnoses were confirmed, follow-up letters were sent and some of the patients were interviewed. There were 64 women in the series and of these 33 were treated by biopsy, simple removal of the tumour mass or a segmental mastectomy. The remaining 31 had simple mastectomies, five of them having bilateral mastectomies.

The pathological findings in the 64 cases were classified as follows:

	Patients
Cystic disease alone	41
Cystic disease with intraductal papilloma	6
Cystic disease with benign neoplasia	8
Cystic disease with fibroadenoma	8
Cystic disease with cystadenoma	1

The follow-up, which was completed during the later months of 1953, revealed 61 patients alive and well with no evidence of malignancy developing in either breast. Three patients had died and from their records and correspondence with their families it was concluded that they died of causes other than carcinoma.

Thus there were 64 cases of proven fibrocystic disease of the breast in which carcinoma of the breast did not develop; for accuracy five of the patients who had bilateral mastectomy might be omitted, leaving 59 who did not develop breast cancer. This finding is in agreement with those of other authors, among whom were Johnson (1924)[11] who reported 101 cases of fibrocystic disease of which 61 were followed up from one to 20 years and no carcinoma occurred.

Bloodgood (1932)[15] reported 350 cases followed up for 10 years and none of these developed malignancy. Campbell (1934),[12] reported 290 patients, two of whom developed carcinoma during the follow-up period; he concluded that cystic disease is not a precancerous lesion and malignant changes are no more likely to develop in a breast showing the disease than is one entirely normal. Lewis and Geschickter[16] after a careful study of 1,048 patients with adenosis or cystic disease reached similar conclusions. A more recent report by Patey and Nurick,[17] in which 65 cases of cystic disease of the breast treated conservatively were followed up from one to 16 years, states that only one patient developed carcinoma.

SUMMARY

An attempt has been made to establish a causal relationship between fibrocystic disease of the breast in all its forms and carcinoma of the breast. The clinical approach has been used.

1. The incidence of previous operations for fibrocystic disease in a series of 313 cases of carcinoma of the female breast proved to be 2.23%. A control series of 300 women of similar age distribution was found to have an incidence of 3% for operations for non-suppurative, non-cancerous lesions of the breast. These series were compared to similar published series.

2. Sixty-four women who had had operations for fibrocystic disease of the breast were followed up over a period of years ranging from eight to 23, the average for the group being 13.15 years. Of this group no patient had died from carcinoma of the breast. Attention has been drawn to the similar results published by other authors.

CONCLUSIONS

1. Patients with carcinoma of the breast are not any more likely to have had previous operations for fibrocystic disease than the normal hospital population of similar age group.

2. Fibrocystic disease of the breast—and this includes fibrocystic disease alone, or with intraductal papillomas or with benign neoplasia or with fibroadenomas—does not seem to develop into carcinoma, at least in our series of 59 patients who were followed up for an average of 13.15 years.

3. Though the follow-up series is not large, the results are in agreement with other published series of larger number of patients with proven fibrocystic disease followed up over a period of years.

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CHANGES IN THE ISO-ANTIBODY CHARACTERISTICS OF GROUP O AND GROUP B DONORS AFTER PROPHYLACTIC VACCINES

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DESPITE EXCELLENT FACILITIES for the cross-matching of recipient and donor bloods in the two largest cities in Alberta, there are numerous occasions on which it is necessary to administer unmatched group O (Rh_0 positive or Rh_0 negative) bloods in emergencies, either because of the urgent nature of the case, or because many smaller hospitals in country districts have inadequate laboratory facilities, and lack qualified technicians.

The heavy demand for "universal donor" blood (transmitted to all hospitals unmatched, but verified as group O Rh_0 positive or Rh_0 negative beforehand) is readily understood when one considers that over 2,700 bottles were administered in this way during 1952, whereas an additional 24,900 bottles of all groups were administered after cross-matching in the two main Canadian Red Cross centres in this province. Since 10% of all blood transfusions administered in this area are with "universal donor" blood, any factors reducing the safety of these unmatched transfusions is of special interest, and we therefore present a further study of the influence of standard Armed Services' inoculations—presently TABTD (TAB, and tetanus and diphtheria prophylactics)—in so far as the natural antibodies are affected by these injections.

Qualitative differences between the natural and "immune" form of anti-A and anti-B agglutinins are now being stressed with greater emphasis, and since the "immune" isoagglutinin is usually associated with a demonstrable hæmolysin, Crawford *et al.*[1] have suggested that the hæmolytic property of the fresh donor's serum could form the basis of a screen test for "universal donor" blood. This group of workers have also demonstrated that TAB, and ATS antitetanic serum frequently contain traces of substance A capable of initiating an "immune" iso-antibody response in a high proportion of individuals thus inoculated.

The members of the M.R.C. Blood Transfusion Research Unit, London (1952) found that of 14 R.A.M.C. officers of group O receiving their initial TAB injections, four of the 14 sera obtained hæmolysed A cells before immunization; and of the 10 officers who had no hæmolysin at all initially, six developed a substantial amount 18 days after immunization. Seven out of the 14 officers showed a significant rise in anti-A titre (fourfold or more). In a few instances these sera also hæmolyzed B cells, particularly after the injection.

The study we present relates to 20 members of the Active Force of the R.C.A.F. who had had previous inoculations of the standard TABT, or TABTD, vaccines, and to a separate group of civilian blood donors.

METHODS USED IN THE TESTS

1. *Hæmolysin tests.*—Group A_1 and A_2 test cells, and the group O "control" cells were obtained from stored donor blood of very recent origin, as were group B test cells. The sera examined were tested, in every possible instance, within two to four hours of collection from the Service and civilian volunteers. The addition of complement from any other source was thus avoided, and reasonably accurate comparisons could be made. Equal volumes of the fresh volunteers' group O or group B sera were mixed with a 5% suspension of the test cells in normal saline, using narrow diameter "agglutinin tubes."

In all cases the 5% cell suspensions plus donors' fresh sera were incubated at 37° C., and examined for hæmolysis at 10 minutes, one hour and two hours' incubation, the two-hour specimens being first gently agitated and centrifuged. The results were recorded as: complete hæmolysis 4 plus, almost complete hæmolysis 3 plus, approximately 50% hæmolysis 2 plus, approximately 20% hæmolysis 1 plus, trace hæmolysis tr or ?, and no trace of pink colour.

2. *Saline and AB serum titrations.*—Serial dilutions of the group O or group B volunteers' sera were made in (a) saline solution, and (b) group AB serum respectively: standard agglutinin or titration tubes were used, the diluted serum in each tube being mixed with a 2% suspension of known test cells of group A_1 or B. The mixtures were examined for agglutination after two hours at room temperature.

3. *"Partial neutralization" with A and B substances.*—For partial neutralization of the volunteers' sera, a saline solution of A and B substances was added to the point where the partially neutralized sera could be made to almost or completely abolish agglutination of A_1 cells in saline. The strength of the A and B substances in saline had to be adjusted for different sera. As Witebsky[2] has demonstrated, relatively large amounts of substances A and B were necessary to neutralize immune antibodies of high titre. Group A_1 cells, and group B test cells were then employed to demonstrate, in typical cases, the characteristic positive indirect Coombs test of the immune sera.

RESULTS OF TESTS

Twenty ml. venous specimens were obtained immediately before and 14 to 18 days after a booster dose of 0.5 ml. TABTD, the findings being compared to those of 22 civilian donors' sera obtained at a regular Canadian Red Cross donor clinic.

TABLE I.

ANTI-A TITRES—SERVICE PERSONNEL
(A_1 test cells and subject's serum)

Subject	*BEFORE TABTD, 0.5 c.c.*		*14 to 18 days AFTER TABTD*		*TABTD Lot No.*	*Past inoculations in R.C.A.F.*
	Saline	*AB serum*	*Saline*	*AB serum*		
1.	1:32	1:16	1:256	1:16,000	Expiry Date Feb. 53 11–1	Tetanus toxoid 1945. TAB 1945.
2.	1:128	1:256	1:128	1:512,000	11–1	TABT, yearly since 1940 TABTD 1952.
3.	1:16	1:16	1:16	1:4,000	11–1	TABT—1945. TABT—1949.
4.	1:1,024	1:16,000	1:256	1:2,000	11–1	TABTD—1952.
5.	1:32	1:64	1:1,024	1:512,000	11–1	TABT—Yearly, 1940 to 1950.
6.	1:32	1:64	1:32	1:64	11–1	TABT—1942. TABT—1943.
7.	1:64	1:512	1:64	1:128,000	Expiry Date Feb. 54 20–1	TABT, and D—1940. TABT, repeated 1941-50.
8.	1:64	1:256	1:256	1:512,000	20–1	TABT—1942. TABT—1943.
9.	1:256	1:256	1:1,024	1:64,000	20–1	TABT—1943. TABT—1951.
10.	1:128	1:2000	1:512	1:64,000	20–1	TABT—1943. TABT—1951.
11.	1:64	1:64	1:64	1:32	20–1	TAB, 1931, 1933. TABT—1948, 1949, 1950.
12.	1:32	1:128				TABT—1940 to 1950. TABT—1951.
13.	1:256	1:256	1:512	1:256	20–1	TABT—1945. TABT—1950.
14.	1:16	1:32	1:16	1:128	Expiry Date Feb. 54 23–1	TABT—1943. TABT—1950.
15.	1:128	1:128	1:64	1:128	23–1	TABT—1941. TABT—1943.
16.	1:64	1:64	1:16	1:128	23–1	TAB, 1929 to 1940. Tetanus toxoid 1938, 1939.
17.	1:32	1:64	1:32	1:2,000	23–1	TAB—1940. Tetanus toxoid—1940. TABT—1941 to 1948.
18.	1:16	1:32	1:16	1:256	23–1	TABT—1951.
19.	1:16	1:64	1:64	1:128,000	23–1	TABT—1940, 1948, 1949 1950, 1951.
20.	1:8	1:4	1:2	1:2	23–1	TABT—1949. TABT—1950.

Of the 20 sera examined before the injection, 10 showed a strong A hæmolysin (+; ++; or more) after 10 minutes' incubation at 37° C., the number increasing to 16 (80%) after one and two hours' incubation, with A_1 test cells. Fourteen of these 20 sera were also hæmolytic to A_2 cells at two hours' incubation, while one serum also strongly hæmolyzed B cells before inoculation. Of the specimens examined 14 to 18 days after the booster dose of TABTD, 17, or approximately 85% were strongly hæmolytic to A_1 cells after two hours' incubation at 37° C., while 15 lysed A_2 cells, and two lysed B cells after two hours' incubation of the test cells and sera.

These results may well be related to the fact that this unselected group of R.C.A.F. officers, N.C.O.'s, and airmen had had similar previous inoculations, the dates of the previous injections being listed briefly in Table I. It will also be noted, from Table I, that many showed a significant increase in anti-A titre against A_1 test cells, particularly when AB serum was used in lieu of a saline medium, before their 0.5 ml. booster dose of TABTD. At least six of the 20 (30%) could be regarded as having a probable immune anti-A in the light of the much higher anti-A titres in AB serum.

Of the specimens tested 14 to 18 days after the booster dose with A_1 cells suspended in saline, and AB serum, there were seven showing

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TABLE II.

INDIRECT COOMBS TEST (ANTI-GLOBULIN TEST)
FOLLOWING PARTIAL NEUTRALIZATION OF SUBJECT'S SERUM

	Indirect Coombs test				*Hæmolysin test*					
	BEFORE TABTD		*AFTER TABTD*		*Comparison with hæmolysin test*					
Subject	*A_1 cells*	*B cells*	*A_1 cells*	*B cells*	*A_1 cells BEFORE TABTD*			*A_1 cells AFTER TABTD*		
					10 min.	*1 hr.*	*2 hr.*	*10 min.*	*1 hr.*	*2 hr.*
1	Neg.	Neg.	Pos.	?	—	—	—	++	++	+++
2	Pos.	Neg.	Pos.	Pos.	+	+++	+++	+	+++	+++
3	Neg.	Neg.	Pos.	Neg.	—	—	+	++	++	+++
4	Pos.	Pos.	Pos.	Pos.	+	+++	+++	+	++	+++
5	Neg.	Neg.	Pos.	Neg.	—	?	tr	+++	++++	++++
6	(?)	Neg.	Pos.	Neg.	—	+	+	—	++	+++
7	Pos.	Pos.	Pos.	Pos.	++	+++	+++	+++	+++	++++
8	(?)	Neg.	(?)	Neg.	+	+	++	+	+	++
9	Pos.	Neg.	Pos.	Neg.	++	+++	+++	++	++	+++
10	Pos.	Neg.	Pos.	Neg.	++	+++	+++	++	+++	+++
11	Neg.	—	Pos.	Neg.	?	++	+++	—	tr	+
12	Neg.	Pos.	/	/	—	+	+	/	/	/
13	Pos.	Neg.	Pos.	Neg.	+	+++	+++	tr	++	+++
14	Neg.	Neg.	(?)	Neg.	?	++	+++	tr	+	++
15	Pos.	Neg.	Pos.	Neg.	(+)	+(+)	++	+	++	+++
16	Pos.	?	Pos.	?	+	+++	++++	+	++	+++
17	(?)	Neg.	Pos.	(?)	?	+	++	—	+	+
18	(?)	Neg.	Neg.	Neg.	=	(+)	tr	—	—	—
19	Pos.	Neg.	Pos.	Neg.	+	++	+++	+++	+++	++++
20	Neg.	Neg.	Neg.	Neg.	—	—	—	—	—	—
Total Positive	9	3	15	3	10	16	16	12	16	17

an increase in titre in the "saline system", but after inoculation, the most remarkable change was detected in the AB "serum system", and where 10 specimens (approximately 50%) had titres of 1:2,000 to 1:512,000 in AB serum.

The volunteers were inoculated with one of three different lot numbers of TABTD (noted in Table I) but it could not be stated that there was any material difference in the results of the tests in relation to the particular lot number of the combined vaccine used. In the remaining specimens there was no noteworthy change, one (No. 4 Table I) actually shows lower titres in both saline and serum after injection. It would seem most reasonable to believe that since one-third of the sera had an increased titre in saline, and one-half had a very greatly increased titre in serum, an A-like substance must be present in TABTD combined vaccine.

In addition to the tests (1) for hæmolysins and (2) of titres in saline and AB serum, it was possible to demonstrate (3) the sensitization of test cells by performing an indirect Coombs test (anti-human globulin test) after partial neutralization of the volunteers' group O or group B sera with Witebsky's A and B substances to the point where the partially neutralized volunteers' sera could no longer agglutinate A_1 cells and B cells in saline.

It was noted that it was particularly difficult to neutralize thus all sera containing high-titre incomplete agglutinins, a detail that tends to strengthen the belief that the addition of standard solutions of A and B substances may not always render such group O blood safe for unmatched recipients of other groups.[3]

In Table II, the findings of the indirect anti-globulin test are compared to the previous results of the hæmolysin test. Before the booster dose of TABTD, nine out of the 20 sera gave a positive indirect Coombs test with A_1 cells (i.e. 45%). Fourteen to 18 days after the 0.5 ml. TABTD injection, the number resulting in positive indirect Coombs tests had increased to 15 (approximately 75%) with A_1 cells—a very marked increase in the percentage of servicemen's sera capable of sensitizing the test cells.

On testing the group O partially neutralized sera with B cells, three were positive before the injection of vaccine, and a fourth group O specimen was also positive 14 days later with the indirect Coombs method. All sera recorded as giving positive indirect Coombs tests had shown strong hæmolytic properties although, as Crawford and others[1] have suggested, the two tests do not necessarily correspond, and variations will be noted.

CIVILIAN DONORS' SERA

While it would be difficult to verify the incidence of immune characteristics in sera derived from a large number of civilian or servicemen's venous blood specimens, we believed that it would be possible to make a limited comparison by performing the same tests on a small number of civilian donors' sera, obtained at a regular Canadian Red Cross clinic.

Accordingly, twenty-two 20 ml. venous blood specimens (19 group O, and three group B) were taken, and these were examined under identical conditions and methods with those obtaining for the 20 R.C.A.F. personnel.

Of the 22 civilians only two were ex-servicemen, both of whom gave a history of inoculations during the final two years of the Second World War. The type of injection could not be confirmed. The remaining 20 male and female volunteers, in a few instances, recalled being inoculated in infancy or childhood, but the histories were too indefinite for any accurate record. The donors were not otherwise questioned, nor were they injected with any vaccines after the specimens were taken at the clinic.

We were greatly impressed by the differences in the results obtained in comparison to those in the R.C.A.F. Active Force group. Five of the fresh sera examined (23%) hæmolyzed A_1 test cell suspensions; two caused hæmolysis of A_2 cells; and none of the group O sera affected B cells. We were most interested to discover that none indicated an intense (++, or greater) lysis of the test cells, both ex-servicemen's sera also being free of any hæmolytic property. We feel that these results offer a remarkable contrast to those of the Active Force Group.

The majority of the anti-A or anti-B titres elicited were low in comparison to those established for the Service personnel, there being but one case in which the titre in AB serum was appreciably higher than in saline. One group O serum had an anti-B titre of 256 (saline), but otherwise no marked variations were noted. After partial neutralization with A and B substances 19 of the sera failed to agglutinate A_1 cells in saline or AB serum; and of the remaining three still possessing a slight agglutinating power in serum two reacted positively on performing an indirect Coombs test.

IMMUNE ANTI-A AND HÆMOLYTIC DISEASE OF THE NEWBORN

Hæmolytic disease of the newborn, although commonly associated with Rh involvement, can sometimes be traced to an immune anti-A response in group O mothers giving birth to affected group A infants. The following results are therefore offered for comparison with those obtained in testing the servicemen's sera. The antenatal patient, Mrs. M.M., is group O, Rh positive: in her last pregnancy the group A infant was severely affected, but no Rh antibodies were detected in her serum. She is now two months pregnant.

The pattern of the "immune" anti-A response, it will be noted, is in keeping with those in the Active Force donors.

Mrs. M.M.'s history is as follows:

Age 36 years: Group O Rh_0 positive (most probable genotype cDE/cde). Husband:—Age 45 years: Group A

1. *Hæmolysin Test*—(Mrs. M.M.'s fresh group O serum):

A_1 cells			*A_2 cells*			*B cells*			*O cells*			*O cells*		
10 min.	1 hr.	2 hr.	10 min.	1 hr.	2 hr.	10 min.	1 hr.	2 hr.	10 min.	1 hr.	2 hr.	10 min.	1 hr.	2 hr.
+	++	++	tr	++	++	—	—	—	—	—	—	—	—	—

II. Anti-A titre (saline) = 1:32
Anti-A titre (AB serum) = 1:128

Anti-B titre (saline) = 1:32
Anti-B titre (AB serum) = 1:16

III. Indirect Coombs test, with patient's partially neutralized group O serum: Positive (A_1 test cells).

Rh_0 positive (most probable genotype cDE/cde). First child, born in 1948, became severely jaundiced within nine hours of birth. The mother's serum, at that time, contained an anti-A, titre 512 (saline). The breast milk specimen on the fifth post-partum day was reported as having an anti-A titre of 128 (saline). Unfortunately there is no report of the Coombs test having been done on the cord specimen at birth. This baby, born on October 15, 1948, recovered within six weeks, after a stormy "hæmolytic crisis."

Mrs. M.M., on questioning, states that she has never had any miscarriages or abortions. Her second confinement was in August 1953, and as of November 1953, the following is a report on her case.

Subsequent tests on the patient's fresh blood specimens showed an increasingly intense hæmolytic action of her serum against A_1, A_2, and finally B test cells. The saline anti-A titre increased to 1:512, and to 1:2048 in AB serum two months before full term.

The infant showed the typical symptoms and signs of a severe erythroblastosis on the day of birth, and the direct Coombs test on the cord blood was positive. An exchange transfusion of 250 c.c. packed cells to which 10 c.c. of A and B substances were added was performed within six hours. This was followed by a steady clinical improvement over the next two weeks, recovery being apparently much more rapid than in the case of her first infant in whose case a replacement transfusion was not attempted.

DISCUSSION

We believe that several British authorities associated with the Medical Research Council Blood Transfusion Research Unit, London, now advocate a standard "screen test" based on the hæmolytic properties of the fresh group O donors' sera. On this broad basis, and bearing in mind the relatively small number of donors tested, we would exclude, as donors of unmatched group O or "bank" blood, 80% of the Active Forces personnel whose specimens were examined before their booster dose of TABTD, against A_1 cell suspensions; and approximately 85% some two weeks after their most recent inoculation. By contrast, of the 19 civilian group O donors only four (or approximately 21%) would thus be excluded as being possibly unsuitable "universal donors" for group A or group AB recipients.

It is of interest to note that the above percentage of civilian group O donors agrees closely with Crawford and others' observation[1] "that the sera of about 20% of group O subjects hæmolyse A cells . . . " Again we recognize the possibility of error in dealing with a relatively small number, although it also happens to agree fairly well with the original observation that about 25% of sera (of all groups) contain isohæmolysin (Moss, 1910[4]).

Since it is impractical to titrate all group O sera in both a saline and an AB serum medium, we would suggest that the detection of A and/or B hæmolysins, as outlined in this paper, could be made the basis of a screen test for the exclusion of potentially dangerous group O donor blood. All group O blood having a potent hæmolytic effect on group A or B test cells could then be reserved for group O recipients only, after crossmatching.

Judging by the high anti-A titres of 2,000 and upwards in AB serum obtained in 11 cases (or about 55%) of those tested within some two to three weeks of inoculation, we would anticipate that the immune response must persist for some considerable time. If we may regard a positive indirect Coombs test as being significant evidence of a certain residual immune anti-A activity, then it would appear that some traces of an immune anti-A response can persist for two years or even longer in a few repeatedly inoculated individuals.

Of the 20 servicemen's specimens examined *before* their current annual inoculation with the combined vaccine, nine (45%) had a positive indirect Coombs test, and of these, seven (35%) received their last TABT, or TABTD injection between 1950 and 1952. Two weeks to 18 days after their booster dose of TABTD, 15 sera (approximately 75%) exhibited a definite positive indirect Coombs result.

Provided donors' sera are available within a few hours of collection, the test for hæmolysins is practical for "blood bank" use, since no additional source of complement is required if this "screen test" can be applied on the same day as the donors attend the clinic. If there is a longer time-lapse than a day, then it would be essential to add fresh serum as a source of complement, necessitating the calling-in of a special donor for this purpose.

Many group O volunteers of the Armed Services are regular blood donors, and we recommend that their blood should always be crossmatched with the recipients' specimens. For an emergency transfusion, and especially where servicemen are the main source of donor blood, we would prefer to administer plasma, or possibly a plasma substitute, pending the matching of the patient's and donor's venous clotted blood specimens, since the screening of sufficient group O's—for unmatched transfusions—will take as long a time as normal cross-matching.

For group O "bank blood", if the civilian donors' sera can be screened within a day of collection, they would be the logical source of "universal donor" emergency transfusions. The

ideal solution is probably the most difficult, namely, the exclusion of all traces of substance A from the "combined vaccine" media.

The question of how long any immune characteristic remains, and the clinical significance of the imune response both in transfusion therapy and in pregnancy is of outstanding importance. As previously stated, we find that the characteristic changes in volunteer group O blood do, in fact, persist for at least two years in a significant number of cases.

SUMMARY

Vaccines such as TABT, or TABTD are believed to contain traces of an A-like substance. After routine inoculations with these vaccines an immune anti-A response, in particular, is likely to occur, making it important to "screen" all group O donors who have received these combined vaccines if their blood donation is to be used, unmatched, for emergency transfusions.

Since the immune isoantibody is commonly found in association with a potent hæmolysin, detection of the latter may be used as the basis of a routine test to exclude potentially dangerous group O blood.

Of 20 servicemen's specimens tested before and after a TABTD booster of 0.5 ml., 80% to 85% showed evidence of a potent hæmolysin, and the majority of these specimens contained an immune anti-A, while a minority possessed an apparently immune anti-B. Over one-third of the Service personnel's specimens, on the basis of the Coombs test, possessed at least a residual immune characteristic, and this immune response may be detected over several years from the last inoculation.

Twenty-two specimens from civilian donors, on the other hand, offered a marked contrast to the Service group results, and we would suggest that unmatched emergency group O blood should be obtained from civilian donors having no recent history of inoculations, reserving the Active Forces personnel for group-specific, matched transfusions only. The hæmolysin test is well worth further consideration as a method of screening fresh group O donors' sera for blood bank use.

The term "universal donor" may sometimes mislead the profession, as it most certainly does not mean that all group O blood is safe even when the Rh positive or Rh negative element is known. Perhaps we should abandon the term universal donor altogether, or at least define its meaning by emphasizing its limitations.

We wish to express our thanks to Colonel E. J. Young, Command Medical Officer, Western Army Command, and his senior colleagues in Ottawa for their kind assistance with previous preliminary studies of this nature; and we are deeply indebted to Squadron Leader L. A. Wright, to Squadron Leader J. W. Stewart and the medical officers at R.C.A.F. Station Edmonton for their great help in making this present study possible.

We were much encouraged to carry out the further tests as a result of the personal interest shown by Dr. R. L. Denton, Montreal, and by Dr. J. S. Cull, Assistant National Director, Canadian Red Cross B.T.S., Headquarters, Toronto.

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Case Reports

CORONARY ARTERY DISEASE IN PREGNANCY

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CORONARY ARTERY DISEASE is a rare complication of pregnancy. It is seldom encountered in the gravid state for reasons which are easily understood. The factors of age and sex are responsible for this situation. It is variously estimated that from only 2 to 5% of all cases of coronary disease develop under the age of 40. Males predominate at all ages but under 40 the difference is very striking with a ratio of one female to 20 males. It is thus evident that we will have to see from 400 to 1,000 patients with coronary disease before encountering the disorder in a young female. Since it may be presumed that even among these few females most will avoid pregnancy, it is evident that this complication will rarely be encountered. The incidence may be estimated from the fact that four of the acceptable cases in the literature were reported from three large clinics, with a total series of

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TABLE I.

SUMMARY OF REPORTED (ACCEPTABLE) CASES, LISTED ACCORDING TO TIME OF INFARCTION IN RELATION TO PREGNANCY

Time of infarction	*Author*	*Year reported*	*Age*	*Previous hypertension*	*Other factors*	*Previous heart failure*	*Termination*
Before pregnancy	Fitzgerald[1]	1951	43	?		Yes	Natural delivery
2 years before	Lyons	1954	37	?		No	Low forceps
1 year before	Horwitz[2]	1943	35	No		No	Cæsarean section
1 year before	Laubach[3]	1951	42	Yes		No	Therapeutic abortion
1 year before	Laubach[3]	1951	41	No		No	Low forceps
At 2 mos.	Brock[4]	1953	34	Yes	Diabetic	No	Low forceps.
2 mos.	White[5]	1937	22	?		No	Low forceps
2 mos.	Hamilton[6]	1941	?	Yes		No	Hysterotomy
4 mos.	Goldberger[7]	1950	37	?		Yes	Delivery 7½ mos.
19 wks.	Stewart[8]	1953	42	Yes		?	Cæsarean
5 mos.	Jensen[9]	1938	39	?		No	Forceps
5 mos.	Mendeson[10]	1952	42	Yes		No	Cæsarean
30 wks.	Mendelson[10]	1952	42	Yes	Syphilitic aortitis	Yes	Died
32 wks.	Stewart[8]	1953	?	Yes		No	Died
Term	Ries[11]	1935	45	Yes		Yes	Low forceps.

about 160,000 maternity cases, or a ratio of one case of coronary disease per 40,000 pregnancies.

A review of the literature disclosed reports of 31 cases, but critical evaluation of these revealed only 14 in which the diagnosis appeared reasonably certain. When a middle-aged male has a history suggestive of anginal attacks plus an abnormal electrocardiogram, the diagnosis of coronary disease may be justified on the basis of probability. In young women, however, other conditions which may produce a similar picture are relatively more common. Myocarditis, pericarditis, metabolic disease, or even neurogenic disorders are more likely to be the cause. More stringent criteria therefore must be applied before the diagnosis of coronary disease can be accepted. An electrocardiogram should be considered diagnostic only if specific changes of infarction are present, viz., pathological Q waves, or reciprocal ST changes with expected evolution in serial tracings. Non-specific abnormalities, such as T wave inversions or conduction defects, may be considered as supporting evidence only if the history is typical of coronary disease and cannot be explained by any condition which may simulate it.

Analysis of 15 cases (14 previously reported together with the case reported herein) reveals the age range to be 35 to 45, with the exception of one patient aged 22 reported by White.[5] Hypertension was present in eight. Of the seven normotensives, in only two was blood pressure known to have been normal before the onset of the disease. Since it is known that the large majority of women who develop coronary disease are either hypertensive or diabetic, it is probable that some of the remaining five patients had elevated pressures before the onset of their disease. One patient had rheumatic heart disease and syphilitic aortitis. The infarct in this case may have been due to syphilitic obstruction of a coronary ostium. There was one diabetic in the group. A history of decompensation prior to the onset of pregnancy was recorded in four patients. Known or presumed infarction occurred before pregnancy in five cases, and during pregnancy in ten. Of these, three developed during the first trimester, four in the second, two in the third, and one at term. There were two deaths, both occurring in the only two patients who suffered infarction in the third trimester (at 30 and 32 weeks). Delivery was natural or by forceps in eight cases; therapeutic abortion was performed in two and Cæsarean section in three. The two patients who died were undelivered.

CASE REPORT

Mrs. L.E., aged 35, was referred to one of us, (B.H.L.) on August 14, 1950 with shortness of breath and chest pain.

The patient stated that one month previously she had been awakened by a painful or "tight" sensation under her sternum. She was unable to lie quietly, but got up, took some alkaline tablets and walked around until relief came in about 20 minutes. Nocturnal attacks of the same character recurred several times in the following month. She also found that when she walked rapidly she experienced a similar discomfort which forced her to stop, whereupon the pain would subside promptly. She stated that she had some shortness of breath on walking half a block. She tired easily, due, she thought, to long hours of work and inadequate sleep. She also

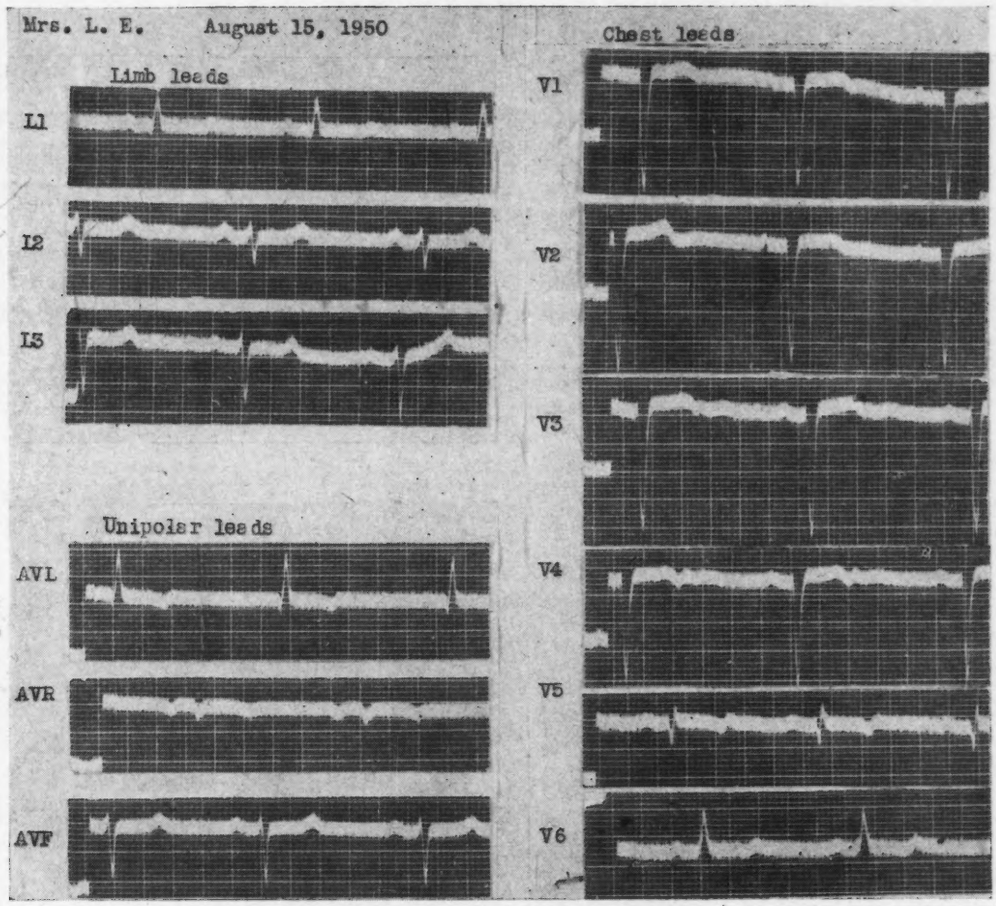

Fig. 1.—Electrocardiogram taken August 15, 1950, following first visit. Sinus rhythm, rate 55. Electrical position horizontal. T waves low in lead one and inverted in AVL. In the chest leads R waves are absent in leads V 1-2, and tiny in V 3-4. A deep wide Q wave is present in V 5. T waves are inverted or diphasic in V 4-5. Interpretation: Anterior (or anteromedial) infarction; age indeterminate.

complained of low back pain and excess menstrual flow for two months.

The patient was born in England. In 1941 (at age 26), she had a thyroidectomy. In 1945 following an uneventful pregnancy and labour she gave birth to her first child. A suspension operation was performed in 1948 to correct a uterine retroversion.

The patient's mother had suffered from angina pectoris.

The essential points in her condition were as follows. A grade 2 sclerosis was noted in the fundi. A thyroidectomy scar was present and no thyroid tissue was palpable. The apex beat was diffuse, the maximum impulse being approximately four inches from the midline. Heart rate was 64 and regular. A slight systolic murmur was heard at the apex. Blood pressure was 112/70. Gynæcological examination (by R.L.) revealed no abnormality except for evidence of previous uterine suspension.

Urinalysis was normal. Sedimentation rate (Westergren) was 20 mm., in one hour; hæmoglobin value 60%, red cell count 3.5 million, and leucocyte count 8,900, differential count and smear were essentially normal. Radiograph of the chest was reported as showing questionable left ventricular enlargement. Basal metabolic rate was minus four. The electrocardiogram disclosed a sinus rhythm, rate 55; electrical position was horizontal. T waves were low in lead one and inverted in AVL. In the chest leads R was absent in V 1-2, and tiny in V 3-4. A deep wide Q wave was present in V 5. T waves were inverted in V 4-5. Interpretation: anterior (or anteromedial) infarction; age indeterminate.

The patient was seen three days later, complaining of further nocturnal chest pain. She was admitted to hospital and placed under anticoagulant treatment. During her hospital stay no significant alterations developed in temperature, pulse, blood pressure, leucocyte count or sedimentation rate. However, serial tracings revealed a changing pattern indicative of active coronary insufficiency. Thus on August 22, lower voltage was present in the limb leads and deep T wave inversions developed in V 1-4. These latter changes had regressed in tracings taken August 25 and September 1, recurred on September 13 and improved again by September 25. On the latter date, however, widening of the QRS complexes to 0.12 sec. was found, indicative of partial or complete left bundle branch block. The block was an inconstant feature in later tracings. Physical examination on September 25 disclosed an increase in the intensity of the apical systolic murmur (from grade one to three), a slight aortic systolic murmur being now present.

After discharge from hospital, she remained fairly well during the summer months, but during the winter she was forced to restrict her activities. She had several severe attacks of pain and electrocardiograms recorded after such attacks revealed varying T wave changes. In 1951 she remarried and became pregnant, the last monthly period being August 30, 1952. During the months of February to April 1953 she had two severe attacks of pain requiring morphine for relief.

On April 24, 1953 the patient came to Winnipeg. The general examination was satisfactory except for the finding of a trace of albumin in the urine and of anæmia. The electrocardiogram was as before with no evidence of recent acute change. The patient was already receiving iron and vitamin preparations. In addition Peritrate was prescribed. After this visit no further chest pain occurred. The authors believe that this improvement was

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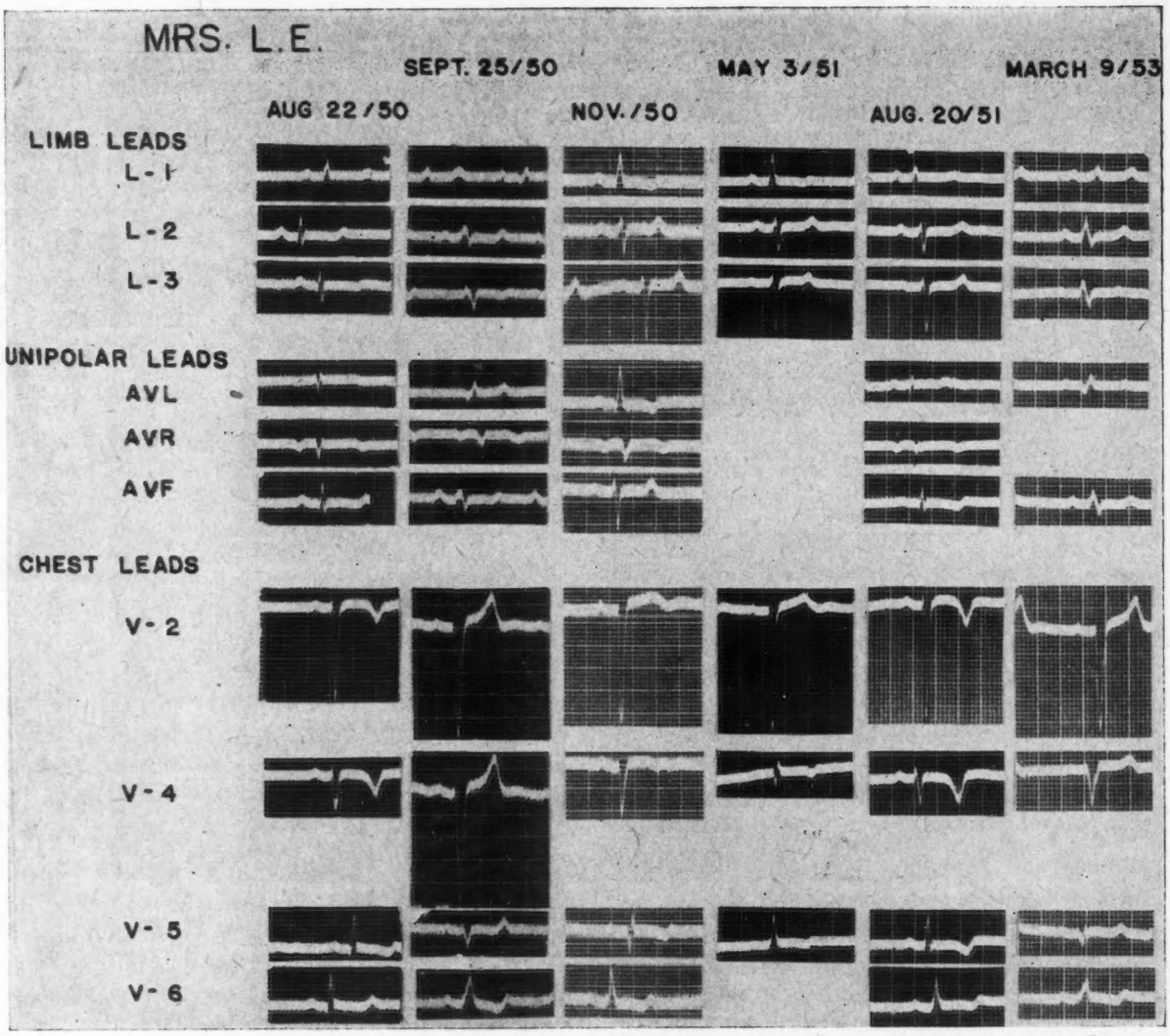

Fig. 2.—Selected electrocardiograms. August 22 taken shortly after admission to hospital shows low voltage with caving of ST segments and deeply inverted T waves in V 1-4. Tracing September 25 (at time of discharge from hospital) shows low voltage and widened QRS—partial or complete left bundle branch block. Subsequent tracings show varying changes during the following two and a half years. Fresh ischæmic changes (T wave inversions) in November 1950, and on August 20, 1951, were taken following acute attacks of pain.

due to the reassurance given, plus the increased rest when away from her home.

On June 3, she was admitted to hospital with recurrent œdema and dyspnœa. Swelling subsided with rest. On June 8 she began to complain of abdominal discomfort attributed to mild hydramnios, and became somewhat anxious and restless. Labour was induced by artificial rupture of the membranes and terminated by outlet forceps under intravenous Pentothal anæsthesia. The post partum course was uneventful and the patient was discharged on the 12th day. Two months later her physician reported her to be doing well.

A blood sample taken two months after delivery was sent to Dr. Gofman of Berkeley, California, who examined the specimen with the ultracentrifuge to determine the distribution of lipoproteins. He reported an athrogenic index of 89, compared with the normal for this age and sex of 41. This change from the normal is commonly associated with the presence of coronary sclerosis.

COMMENT

In recent years intensive studies of heart disease in pregnancy have provided generally accepted guides to prognosis and management. It is known that pregnancy imposes an additional load on the heart, presumably because of increased blood volume. This attains its maximum at about 32 to 36 weeks and then declines. It has been found that the important consideration in prognosis is not the nature of the lesion, but the functional capacity of the heart as manifested by such symptoms as impaired exercise tolerance or dyspnœa. Further evidence that the disease is progressive or has reached an advanced stage may include such signs as increasing cardiac enlargement or development of arrhythmias. With the cardiac reserve already depleted, it may be feared that the extra load of pregnancy will not be well tolerated.

Such factors as the age of the patient, ability to secure help in her work, parity and anxiety for a child are all to be considered. During pregnancy close supervision is mandatory. Adequate rest periods must be insisted on; exertion which causes distress should be avoided; infections must receive immediate attention. Severe hypertension poses a special problem because the incidence of toxæmia and fetal death is greatly increased. Should failure develop during early pregnancy no interference is permissible before maximum compensation has been attained. When failure occurs in the latter months of pregnancy the heart may be "coaxed along" in the hope of getting the patient "over the hump" of high blood volume at the 36th week. For delivery it has been conclusively proved that the vaginal route is the method of choice, Cæsarean section being reserved for obstetric indications. Analgesia in the first stages, and easing of the second stage by caudal or other anæsthesia, plus episiotomy and low forceps, are accepted methods of treatment. In cases of coronary disease it would appear wise to avoid ergot and pituitary preparations, since these are known to be capable of inducing coronary insufficiency.

The management of coronary disease poses a special problem. Some or possibly a majority of patients encountered will be advised against pregnancy because they fall into the group with unfavourable prognosis, either because of poor functional status with diminished cardiac reserve and early signs of failure, or because of severe hypertension which is usual in women with coronary disease. However, the rarity of reported cases has prevented a conclusive ruling on management of the patient with coronary disease but good cardiac functional capacity, the heart little enlarged, and hypertension absent or mild. This was the problem encountered in the case reported above.

In reviewing the 15 reported cases one point which appears significant may be noted. All patients whose infarction occurred prior to the third trimester of pregnancy and who continued with the pregnancy carried it through successfully. This would suggest that there is some mechanism increasing coronary blood flow during pregnancy to meet the increased demands of the heart during the third trimester. It is to be noted on the other hand that the two patients who did develop infarction in the third trimester of pregnancy died. This suggests that an acute infarction developing during this period of high blood volume is not well tolerated, whereas a previously damaged heart may adjust itself to the increased load. While the number of cases under consideration is small, this conclusion is probably statistically significant (less than 2% probability of this finding occurring by chance).

In view of the scanty information available ordinary prudence would suggest that the patient with coronary disease be advised against pregnancy, since there is no means of foreseeing development of infarction at an unfavourable time (third trimester). However, given a pregnant patient with coronary disease whose functional status is not unfavourable, and who is anxious to continue with her pregnancy, it is justifiable to let pregnancy go on, providing constant supervision and full co-operation can be maintained. The general principles enumerated in the care of heart disease apply. The use of anticoagulants during the last trimester of pregnancy should be considered in those patients who have anginal symptoms during this period.

SUMMARY

Coronary disease in pregnancy is a rare event. Fourteen acceptable cases have been found in the literature, and a further case is reported of a patient with coronary disease who successfully carried through a pregnancy.

Of the total of 15 reported patients, 13 survived. The two patients who developed infarction in the third trimester died. It is suggested that it is at this period that infarction is likely to be fatal.

While available information on which to base opinions is scanty, it is suggested that pregnancy be avoided in the presence of coronary disease. If the patient is already pregnant, and there are no other contraindications, it may be justifiable to try to carry the patient along with great care and vigilance.

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A CASE OF RHEUMATIC HEART DISEASE WITH LONG-TERM USE OF ANTICOAGULANTS*

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CHRONIC RHEUMATIC heart disease is an important cause of embolic episodes.[1] These usually occur during chronic auricular fibrillation in patients who are on a maintenance dosage of digitalis. Daley *et al.*[2] found that approximately 50% of all emboli were cerebral in location and caused a mortality of about 50%. In 194 cases studied, 30% died following a single embolic episode. 48% of the patients who survived the first embolization succumbed to subsequent ones.

Encouraging results have been obtained with the long-term use of anticoagulants in the prevention of recurrent embolic episodes.[3 to 6] The following case features the successful use of an anticoagulant for approximately one year, in a woman with rheumatic heart disease who had previously suffered several embolic episodes. It also emphasizes the prompt treatment of a bleeding episode with vitamin K_1.

A 36 year old woman was first admitted to the Reddy Memorial Hospital in October 1951 for evaluation of her cardiac status. After appropriate clinical, electrocardiographic and fluoroscopic studies, a diagnosis of rheumatic heart disease with cardiac enlargement, mitral stenosis and insufficiency and auricular fibrillation, was made. The patient was categorized as decompensated Class 2 (c) and, following digitalization, was placed on a low salt diet and referred to the Out-Patient Department for periodic mercurial injections.

In February of 1952 she was admitted to another hospital with the sudden onset of chest pain and hæmoptysis of bright red blood. After a stormy course she recovered and was discharged home.

In December of 1952 she was readmitted to the Reddy Memorial Hospital. Two weeks ago she noted the sudden onset of numbness of the left side of the face, left arm and left leg, lasting only two minutes and leaving no residual symptoms. On the night prior to admission she had a similar attack in which only the left arm was involved. Three more such episodes occurred during the first few days of hospital stay. During one of these "attacks" numbness of the right arm lasting a few minutes first occurred, followed in ten minutes by transient numbness of the left side of the face, tongue and left arm which persisted for five minutes. A medical man was unable to reach her quickly enough during these episodes in order to examine her. Neurological examination subsequently showed nothing abnormal.

It was considered that the patient was having multiple small cerebral emboli and that in February 1952 she had had a pulmonary infarction. A decision to use anticoagulants was made in view of the grave danger that a subsequent embolus might be larger and perhaps fatal.

Cognizant of the possible dangers which might ensue from the use of anticoagulants during the acute phase of cerebral embolization a two week waiting period was enforced and dicoumarol was then started. The prothrombin time prior to therapy was 16 seconds (normal 15 seconds). Daily prothrombin time determinations were made using the principles of the one stage method of Quick.[7] It was found that by giving the patient 50 mgm. of dicoumarol daily, as a maintenance dose (after initially reaching a prothrombin time of two or two and one-half times the normal), the prothrombin time could generally be kept at a satisfactory level. (See Discussion).

Following discharge she returned to the laboratory three times weekly for prothrombin time determination. Subsequently this was decreased to twice weekly and then to once every seven to 10 days. If the level rose above two and one-half times the normal, the patient was advised to omit her dicoumarol for a day or two and then reduce it to 25 mgm. for a few days. On the other hand if the prothrombin time fell below 22 seconds (in the presence of a control of 15 seconds) she increased the dosage to 75 mgm. daily for a few days, resuming her maintenance dose subsequently. Only very few such adjustments were necessary and in each case one of the authors was consulted and advised the patient. Prothrombin time determinations were checked more frequently during such episodes. In this way the patient's coagulation level has been maintained satisfactorily during the past year. She has had no further embolic episodes.

However, after six months of therapy and three days prior to her normal menstruation she noted profuse vaginal bleeding. Inasmuch as she had been warned about bleeding she immediately advised the hospital and was admitted. The physical findings were similar to those present on the first admission. The blood pressure was maintained at 130/80. The hæmoglobin level was 80% compared to 92% during her first admission. The prothrombin time was 85 seconds at 6.00 p.m. on the day of admission. (Five days earlier it had been 26 seconds). At 7.00 p.m. the patient was given 50 mgm. of vitamin K_1 (Mephyton, Merck) intravenously, slowly. At 11.00 p.m. that night the prothrombin time was down to 60 seconds. At 8.00 a.m. the following morning the prothrombin time was 25 seconds. During the night the bleeding subsided and fresh whole blood which had been available did not have to be used. Subsequent prothrombin time values on May 11, 12 and 13 were 24, 21 and 19 seconds respectively. The patient was restarted on dicoumarol and returned to her previous maintenance dosage of 50 mgm. per day. She has continued on this regimen to date and has had no further trouble. It is hoped shortly to lengthen the intervals between prothrombin time determinations to twice monthly.

DISCUSSION

The chief problem in any long-range prophylactic use of anticoagulants is the maintenance of an adequate reduction in blood coagulability while simultaneously avoiding the dangers of bleeding. Thus Griffith *et al.*[8] found a striking reduction in the incidence of thromboembolism in patients treated with the coumarin anticoagu-

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lants who were maintained with prothrombin times of less than 60% of normal. Brambel[9] found that protection appears to be offered when anticoagulants are used prophylactically if the prothrombin level is maintained at 40% to 50% of normal. Cosgriff[10] kept 28 patients with recurring embolism on prophylactic anticoagulant therapy with a range of prothrombin times of 30% to 15% of normal. With this programme he found that 103 emboli had occurred during 275 patient-months prior to treatment, compared with 13 emboli during 625 patient-months under therapy. Furthermore approximately three-quarters of the 17 patients in whom long-term use of anticoagulants had been discontinued suffered another embolism.

On the basis of the above figures our patient has been maintained continuously within a successful "therapeutic" range. In only four instances was the prothrombin level greater than a value corresponding to 30% of normal prothrombin activity, and even these four determinations represented values below the range of 40% to 60% of normal considered effective by the above-mentioned authors in the prophylaxis of thromboembolism. The obvious principle that therapeutic success with lessened danger of bleeding could be obtained by aiming at a relatively lesser prothrombin reduction was, therefore, adhered to in the case under consideration. The one bleeding episode which occurred with a markedly prolonged prothrombin time did so without any apparent precipitating cause. Fortunately with the availability of fat soluble vitamin K preparations such situations can be fairly quickly reversed.

It should also be noted that long-range studies (two days to 56 months) by Meitus *et al.*[11] in 45 patients have revealed no evidence of appreciable hepatic parenchymal damage in patients on dicoumarol who had not previously suffered from liver disease.

SUMMARY

Another case is added to the growing list of patients with intracardiac thrombosis and recurring embolization who have been kept on continuous anticoagulant therapy with a significant reduction in further embolic episodes. In this instance, a 36 year old woman has been successfully maintained on dicoumarol for 11 months with no further recurrence of emboli. One bleeding episode was successfully treated with vitamin K_1.

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ADDENDUM:—A total of 18 months has now elapsed since the onset of treatment. The patient continues at a favourable prothrombin level without further embolization or bleeding.

FANCONI'S ANÆMIA (APLASTIC ANÆMIA WITH CONGENITAL ABNORMALITIES)*

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TWENTY-THREE CASES of aplastic anæmia with congenital defects—described first by Fanconi—have been published to date. We have recently followed up a further patient who had two siblings affected by this syndrome, which, so far as we know, has hitherto not been described in Canadian medical literature.

The parents of the child (both of Ukrainian origin but not consanguineous) came to Canada 30 years ago. The oldest brother, born in 1927, is alive and well. The second brother, born in 1928, had small thumbs on both hands. He died three days after birth. The next three siblings—a brother 23 years, sister 19 years and brother 15 years—are apparently normal. The next child, a girl, was born in 1942. She had no thumbs; her forearms were shorter than normal, and both hands were flexed radially. Radiographs taken at the age of four showed absence of both radial bones and delayed ossification centres of the right carpal region. This girl was noted to have darker skin than the other siblings. At the age of nine, she developed measles with a typical rash, and five days from the onset of the measles she was admitted to a country hospital because of nasal and rectal hæmorrhage, fever and pneumonia. Her hæmoglobin value on admission to hospital at that time was 22%. Blood transfusions and antibiotic therapy did not achieve improvement, and the patient was discharged home, when she died in June of 1951. (These data were kindly furnished by Dr. M. E. Chonko, Two Hills, Alberta.)

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*Not to be confused with other "renal" Fanconi's syndrome.

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The patient here reported was the youngest child in the family, and was nine years of age when seen by us. Pregnancy and delivery in her case had been normal, and the child is alleged to have developed at a normal rate and to have been making satisfactory progress at school. During the past two years, she had frequent nose bleeds, bruised easily, and complained of weakness and nervousness. Six months prior to admission to this hospital she had a severe epistaxis, since which she has been unable to attend school. She was sent to this hospital on April 24, 1953 for investigation.

Fig. 1.—Patient (right) and normal girl of her age.

On examination, her stature was found to be smaller than average for her age (Fig. 1). Her weight was 50 pounds. The skin was uniformly dark with olive tint. No petechiæ or bruises were present. The head was smaller than normal, and microphthalmia was noted. The occipito-frontal diameter on lateral radiographs of skull was 17 cm., as compared with 20 cm. and 21 cm. in two normal girls of the same age. The right thumb was much smaller than normal and attached loosely only by the skin. It could be moved passively in all directions but no active movements were possible. The left thumb was smaller than normal, and in form more similar to the other fingers than to a thumb (Fig. 2). No other bony deformities were detected. The ears did not show visible abnormalities, but hearing was grossly impaired on both sides. The girl did not hear when spoken to in a low voice, but stated that she heard the tuning fork from a distance too great to be possible. The fundi showed normal discs without physiological cupping. In the right fundus, two large hæmorrhages radiating from the disc towards the periphery were seen. The chest was clear. A soft, grade 1 systolic murmur was heard over the apex and in the second intercostal space, both to the left and right of the sternum. The liver was palpable just below the costal margin. The spleen was not palpable.

Reflexes.—Knees—markedly hyperactive; ankles, biceps, triceps, and supinator—slightly hyperactive. No true clonus. No other neurological abnormalities were detected. The child's intelligence appeared normal but rapport was difficult to establish because of her deafness. Bone marrow was aspirated from the iliac crest, and smears showed a very acellular marrow giving a differential of:

	%
Neutrophil polymorphs	7
Neutrophil stab cells	5
Neutrophil metamyelocytes	1
Lymphocytes	50
Acytoplasmic nuclei	24
Eosinophil polymorphs	1
Reticulum cells	3
Unidentified cells	1
Intermediate normoblasts	1
Late normoblasts	7

No megakaryocytes or megaloblasts were seen in the smears of marrow, in which numerous crystalline and amorphous clear bodies were seen and later shown to be fatty acid crystals. Other laboratory findings are given in the table.

Skin biopsy from the abdominal wall showed a marked increase in the amount of pigment in the basal and lower prickle cell layers together with free pigment in the upper corium and also in large melanophores in the same area. Special staining for iron gave negative results, and it was presumed that the brownish pigment was of melanin nature.

Chest radiographs were normal. Those of the right hand showed under-development of the bones of the right thumb and first metacarpal bone with a thin radius, and six carpal ossification centres (normal for her age eight). Radiographs of the left hand showed a short last phalanx of thumb, and seven carpal ossification centres (Fig. 3). The feet were moderately decalcified but otherwise normal. The radiograph of the skull was normal, with a normal sella turcica. Intravenous pyelography showed normal kidneys.

During her nine-day stay in hospital, the patient ran a temperature of from 98 to 100° F. She received seven blood transfusions, each of 250 ml., without any reaction. After five transfusions, her hæmoglobin value rose to 8.6 gm. (59%) and her erythrocyte count to 2.9 million, while her platelet count appeared to rise to 90,000. The patient was discharged on May 3, 1953, and the parents were advised to take her to her country hospital for

TABLE OF LABORATORY FINDINGS

Peripheral blood	Red cell count 1.2 million: Hb 3.5 gm. (24%); M.C.H. 29.1.
White cells	4,500 per c.mm.; polymorph 30%; lymphocytes 65%; monocytes 4%; eosinophils 1%; normoblasts 1%; reticulocytes 0.5%; platelets 60,000 per c.mm. Anisocytosis—slight. Schistocytes present; blood group B, Rh-negative.
Coagulation	clotting 9½ mins. (Lee-White); prothrombin time 16 sec. (80%).
Coombs test	Negative.
Red cell fragility	Hæmolysis began in 0.45% and was complete in 0.3% saline.
Capillary fragility	No petechiæ (5 min. at 80 mm. Hg.).
Serum bilirubin	Total 0.25 mg. %.
Serum protein	Total 6.9. gm.; albumin 3.4 gm.; globulin 3.5 gm. %.
Urinalysis	Negative. No porphyrins or porphobilinogen present.
Blood sugar	Fasting 100 mg. %; 3 hr. p.c. 125 mgm. %.
Thorn test	Eosinophils at 8 a.m. 69 per c.mm.; at 12 noon 103 per c.mm. Uric acid/creatinine ratio: 1st specimen 82.1/136.3 = 0.6; 2nd specimen 44.3/36.3 = 1.22.
Kepler-Power test	Night specimen 100 ml.; 8.30 a.m. 58 ml.; 9.30 a.m. 42 ml.; 10.30 a.m. 124 ml.; 11.30 a.m. 23 ml.; 12 noon 20 ml.
Serum sodium	161 meq/l. (uranyl zinc acetate method—not checked).
Serum chloride	106.1 meq/l.

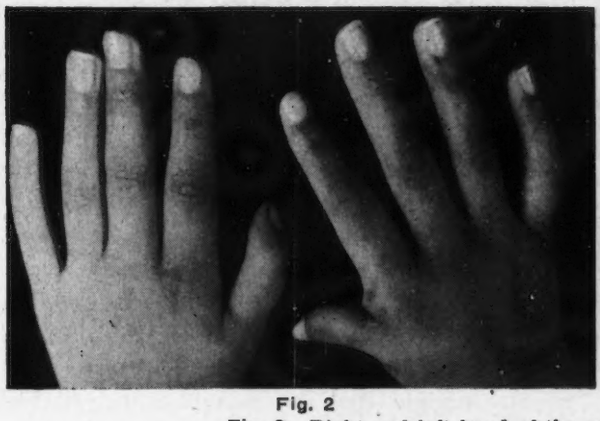

Fig. 2

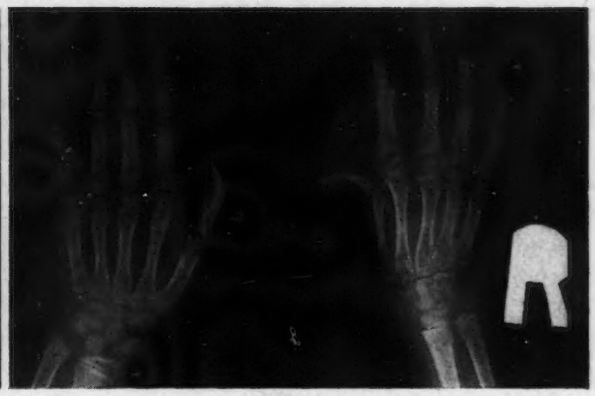

Fig. 3

Fig. 2.—Right and left hand of the patient. Fig. 3.—X-ray of both hands.

more blood transfusions. One week after returning home, nose bleeds again set in and nasal packing was required. Her hæmoglobin value just after the onset of the nasal hæmorrhage was 30%. Several blood transfusions were required as her epistaxis increased. She ran a slight fever despite antibiotic therapy. On May 20, the patient died at home. No autopsy was performed.

DISCUSSION

The diagnosis was certain in our case. Aplastic anæmia with leukopenia, thrombocytopenia, congenital defects of thumbs, microcephaly, deafness, microphthalmia, dark colour of skin, exaggerated tendon reflexes, familial incidence – all these fit perfectly into the condition first described by Fanconi.[1] Since Fanconi's publication, only a few cases of familial incidence of this syndrome have been described (Weil,[2] Hjorth,[3] Dacie and Gilpin,[4] Rohr,[5] Levy,[6] Kunz[7]). Several sporadically occurring cases have been reported, by Uehlinger,[8] Van Leeuwen,[9] Weil,[2] Zellweger and Zollinger,[10] Estren, Suess and Dameshek,[11] Diamond,[12] Beautyman,[13] Baumann,[14] Young *et al.*,[15] and Silver, Blair and Kempe.[16] An exhaustive review of the literature was recently published by Kunz.[7]

Elevation of the reticulocyte count and abnormal fragility of the red blood cells were present in six reported cases, and elevation of serum bilirubin level was noted in five cases, but all these findings were absent in our patient. Macrocytosis and the presence of schistocytes were noted in our case, but anisocytosis was slight.

Kunz suggested impaired adrenal function as the cause of some of the manifestations of this syndrome. His view was based on the findings by Gasser and Hollander (Kunz[7]) of a low kestosteroid excretion in the second case of Rohr, and a positive Kepler test and suggestive changes in serum electrolytes in the same case. In our case, however, Kepler and Thorn tests were normal and the serum sodium value was high. We agree with Kunz as regards the striking resemblance of all these patients to each other in the published photographs. Our patient resembles very closely the photograph given by Beautyman.

Out of 26 patients reported to date, (including the three here described), only one recovered, this patient being without congenital defects (Dacie and Gilpin[4]), and two are alive (Rohr and Beautyman, cited by Kunz). As far as treatment is concerned, only blood transfusions and antibiotics remain of certain value in prolonging life. Splenectomy seemed helpful in the case of Dacie and Gilpin, and improved initially the patient of Estren *et al.*, who, however, died subsequently (Kunz). In other cases no change in the course of the disease was noted. Gasser and Hollander used cortisone unsuccessfully in two cases (Kunz).

It seems to us that the syndrome above described is not as rare as might be judged from the small number of reported cases. Probably, in many cases the diagnosis is missed. It is characteristic that several cases were published from Zürich,[1, 5, 8, 10] and a number of the remainder from big centres in the United States. The full description of the syndrome has never appeared in general journals in the English language, and may well be unknown to many practitioners and orthopædic surgeons who may have occasion to see such cases. Probably also, many more patients with multiple congenital defects do not live long enough for the development of the characteristic anæmia, which usually appears around the age of seven years. In such patients, proper diagnosis must remain presumptive. As an example of such a case, we may mention a three day old baby recently brought to our attention

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by Dr. M. Milner. This child had a marked radial deviation of the left hand due to absence of the radius, the left thumb being also absent. The right thumb was like a second finger and was attached only by skin, while the radius on the right side was hypoplastic and the hand radially flexed on that side. In addition, this baby showed multiple anomalies of the vertebræ, mostly in the form of hemivertebræ. The hæmatological picture was normal: erythrocyte count 6.4 m., Hb 18.4 gm. (127%), white cell count 10,600, polymorphs 40%, lymphocytes 46%, monocytes 10%, eosinophils 4%, platelets 500,000.

Fanconi's syndrome may have an importance beyond its rarity in that it may help to explain the etiology of other aplastic anæmias without toxic causes on the basis of a congenital defect of bone marrow. If this syndrome may occur with one or more congenital defects, it is conceivable also that in some cases the only defect may be that affecting the marrow. Eight such cases in two families have been described by Estren and Dameshek,[17] and one whose sibling had anæmia and congenital defects by Dacie and Gilpin.[4]

SUMMARY

Three cases of aplastic anæmia with congenital defects (Fanconi's anæmia) of familial occurrence are presented. The possible connection of this syndrome with idiopathic aplastic anæmias is briefly discussed.

Our thanks are due to Mr. Z. A. Zielinski, Photographer, Royal Alexandra Hospital, Edmonton, for the photographs.

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MALIGNANT THYMOMA*

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MALIGNANT THYMOMA is fortunately a fairly rare condition. According to Boyd[1] "Malignant thymoma resembles in structure a lymphosarcoma, but careful examination will reveal a difference. The cells are larger than those of the typical lymphosarcoma, large pale cells recalling those of Hodgkin's disease may be seen, and occasionally giant cells are present. Although it is commonly thought that the cells are derived from the lymphoid cells of the gland, Ewing and others consider that they originate from the reticulum cells." These tumours "compress and invade the surrounding structures, and may extend downward as far as the diaphragm. The bronchial, cervical and axillary lymph nodes may become involved. Thymoma forms a variety of mediastinal tumour. Metastasis to distant organs sometimes occurs."

Both Bell[2] and Moore[3] mention thymoma but do not add a great deal to the general picture. Rider and McDonald[4] and also H. Levine[5] mention malignant thymoma in reports published in 1950.

I wish to report a case which I saw recently.

CASE HISTORY

E.G., a girl of 13, was referred to me on March 26, 1953, with a history of having been treated for enlarged glands in the neck five days previously. She was given large doses of penicillin and sulphonamides without relief and the glands continued to enlarge.

On the morning of March 26 the mother informed the family doctor that her child had had several very severe episodes of choking, difficulty in breathing and cyanosis. The mother took the child to the local hospital where she was given oxygen periodically. The family doctor saw her in one of these attacks and felt quite concerned. He thought that he might be dealing with a Ludwig's angina with a possible acute œdema of the glottis.

I saw her about 8 p.m. on March 26. She appeared to be a very happy teen-ager without any distress. There was tremendous swelling of the neck reaching from the submental area to the supraclavicular notch. This mass seemed to be fairly soft but there appeared to be hard lymph nodes at both outer margins in the cervical region.

I thought we might be dealing with a Ludwig's angina or a thyroiditis, but the patient had no voice changes whatever. She had no difficulty with her tongue. The larynx showed no evidence of acute œdema. In view of the history of the case, we warned the mother that we might have to do a tracheotomy during the night. Aureomycin intravenously was ordered despite the fact that she had no temperature. The child had a good night but in the morning she suddenly clutched her throat, became very cyanosed, leaped out of bed and collapsed at the door of her room. By the time the nurse got her

*Presented at the June 1953 meeting of the Canadian Otolaryngological Society at Minaki Lodge.

into bed, the child was deeply cyanosed and appeared to be dead. A soft intratracheal catheter was passed and artificial respiration with oxygen was applied, but without response.

The autopsy report in summary was as follows. The body was that of an overly-developed teen-age girl, looking more like 16 than 13. There was an extreme degree of cyanosis, deeper and more diffuse than is usual in asphyxial deaths. The sternum bulged forward in the upper third as if it were being pushed outward by something in the upper thorax and imparting a "pigeon-breast" shape to the chest. The soft tissues of the neck and upper thorax were wet and œdematous though the veins were not engorged. On removal of the sternum a large mass was found in the region of the thymus gland. This mass weighed 660 gm. and measured 7 x 5.5 x 9.5 cm. and was of a mottled greyish-white colour and soft consistency. The central portion was traversed by fibrous trabeculæ producing a lobulated pattern. It was adherent to the anterior superior pericardium and surrounded the great vessels. It did not extend above the cricoid cartilage. Posteriorly it compressed the trachea. The thyroid was not involved and appeared normal, though metastatic deposits were present in both the cervical and mediastinal lymph nodes. Smaller metastases were also demonstrated in the liver, kidneys, left ovary and uterus. Microscopically all the tumours proved to be of a similar type, consisting predominantly of variegated small round or ovoid hyperchromatic cells with scanty cytoplasm. Hassall's corpuscles could still be identified in sections taken from the primary mediastinal growth. On the basis of these observations, a diagnosis of thymoma of the lymphosarcoma type was made.

The extreme degree of cyanosis is emphasized as being out of proportion to the degree of mechanical tracheal obstruction. This phenomenon is consistent with certain observations made by Schwartz[6] who is studying the effects of thymic extracts on man and animals. Extracts have been obtained by which a state of shock can be induced. characterized by bradycardia and profound cyanosis.

In view of this it is questionable whether tracheotomy would have been of help in this case, since in addition to bronchial tree obstruction it is possible that the neoplasm may have been functionally active and elaborating toxic substances analogous to the extract recovered by Schwartz. It is also pointed out that radiation therapy would have been of questionable value because of the rapidity of the fatal episode.

Further information was obtained from the mother that may or may not be pertinent. This child had always been "sickly" and was thin, asthenic and underdeveloped up until about a year before death, when she suddenly began to blossom into premature maturity with a gain in weight, development of secondary sex characteristics and a general increase in growth. Menses also appeared at about this time and the patient showed an increase both in energy and in interest in her environment.

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AN ATYPICAL FORM OF DE QUERVAIN'S DISEASE

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SINCE 1895, painful stenosing tenovaginitis of the abductor pollicis longus and extensor pollicis brevis at the radial styloid has been described repeatedly under the name of de Quervain's disease. It is characterized by pain over the radial aspect of the wrist, which is aggravated by ulnar deviation of the hand; abduction of the thumb is usually impaired and there is sometimes weakness of the grip or even sudden dropping of objects from the hand. A slight swelling in the styloid area and along the tendons is common.

It was always assumed that locking on movement or snapping did not occur in this syndrome, and this has never been reported. Lapidus[3] did not see it once in 165 cases, although the observation recorded below shows that it is a possibility.

A 19 year old student nurse had complained of pain in the radial aspect of the left wrist for about six months and could rely only on her right hand because of weakness and insecurity in the left. Examination revealed some tenderness and swelling over the left styloid process, and the patient was only able to abduct the thumb to 40 degrees, compared with 60 degrees on the right. With the thumb in the abducted position the terminal phalanx was slightly flexed and could be further flexed, but adduction and flexion at the metacarpo-phalangeal and the carpo-metacarpal joints were impossible. The patient could disengage the locked tendons by pushing on them where they form the radial border of the anatomical snuff box distal to the styloid process. Another method of obtaining the same result was by bending the thumb with the other hand, adduction occurring with a sudden recoil accompanied by a click. This procedure could be repeated but became more and more painful with each repetition.

An operation was performed under local anæsthesia. The tendons were exposed through a transverse incision, half an inch proximal to the main crease of the wrist. The radial retinaculum for the abductor pollicis longus and the extensor pollicis brevis were found to be normal in appearance but a stenosis was suspected at its distal third because of the snapping which could be felt on

movements of the thumb. The fibrous sheath over the extensor pollicis brevis was slit and a ring-shaped thickening 4 mm. wide was seen on the inside. However, the patient could still feel some resistance on adduction and it was felt that the fibrous sheath over the abductor pollicis longus should also be opened; this was done and movements became free instantaneously. Not until then was it noted that the sheath of these two tendons, which is usually common to both, was divided by a strong, thick septum, rendering the two tendons completely independent of each other (Fig. 1).

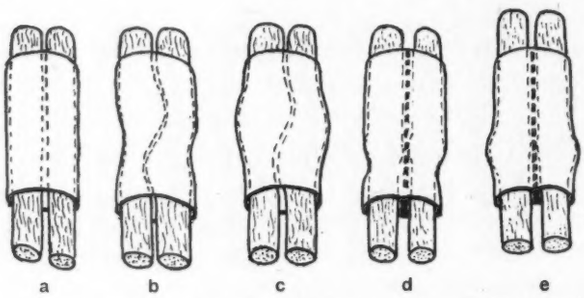

Fig. 1.—Illustration of the relation between ext. poll. long. and abd. poll. brev. and its fibrous sheath under normal condition (a), in de Quervain's disease without locking (b, c), and in de Quervain's disease with locking due to separate sheaths (d, e).

Movements of the thumb and hand were encouraged immediately after the operation, the patient resuming her ward duty the following day. There was no pain and she could use the left hand as well as the right after four days.

A knowledge of anatomy will show why locking is so frequently found in stenosing tenovaginitis of the flexor tendons, but not of the tendons around the styloid. Each flexor tendon possesses its own fibrous flexor sheath, and if the sheath is narrow or the tendon thickened no means of by-passing this obstacle is available. The extensor retinaculum, on the contrary, forms six compartments by attachment of septal bands to the distal ends of radius and ulna. The radial compartment serves for transmission of two tendons, the abductor pollicis longus and the extensor pollicis brevis. If there is a narrowing of the sheath or if there is nodular thickening of one or both tendons, the swollen portion will simply compress the neighbouring tendon. It may then distend the sheath uniformly, thus producing pain, but it will not lead to locking. On the other hand, if the compartment is divided by a fibrous septum into separate sheaths, the mechanism seen in the flexor tendons comes into play.

So rare is the presence of a separate sheath that absence of locking and snapping is almost considered a *sine qua non* for diagnosis of the condition. The latter assumption would place the case described in the broad group of stenosing tenovaginitis either in the sub-group of snapping fingers or as a special type of de Quervain's disease.

SUMMARY

A case of de Quervain's stenosing tenovaginitis with the symptom of locking and snapping is reported. The rare occurrence of this symptom is explained on the basis of an anatomical abnormality.

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ACCIDENTAL MESANTOIN POISONING IN AN INFANT*

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ACUTE MESANTOIN (3-methyl-5-ethyl-5-phenylhydantoin) poisoning is rare. The only known cases reported are those of Kozol (1946) and Savoy (1946). Their patients were young adults. The following report describes the sequence of events occurring in an infant who swallowed 1.5 gm. of mesantoin within a period of 15 minutes.

The patient was a female infant, 19 months of age, who weighed 27 lb. Her birth and early infancy were normal. At seven months of age she had generalized convulsions following inoculation with a combined tetanus, diphtheria, and pertussis vaccine. Thereafter she had recurrent convulsions at intervals of a few weeks until the age of 11 months. At this time she was admitted to hospital for investigation. There was no family history of epilepsy. Physical examination, urinalysis, examination of cerebrospinal fluid, blood Wassermann testing, and pneumo-encephalography revealed no abnormality. Treatment with phenobarbitone and later dilantin failed to control the convulsions. In the month preceding the admission for acute poisoning she was treated with mesantoin 0.045 gm. t.i.d. and was free of convulsions.

Between 10.30 and 10.45 a.m. on November 13, 1952, she swallowed 15 tablets of mesantoin, a total of 1.5 gm. On admission to hospital at 11.00 a.m. she was very drowsy, but responsive to painful stimuli. The pupils were contracted but reacted sluggishly to light. The arm and leg reflexes were diminished. Otherwise the physical findings were normal. Immediately after admission her stomach was washed out with tap water. A small quantity of powdery material was present in the first washing. The final washings were clear.

At 12 noon the infant was difficult to arouse. Caffeine sodium benzoate (60 mgm.) was administered intramuscularly. Ten minutes later she could not be aroused. Picrotoxin (3 mgm.) was administered intramuscularly, but there was no apparent response. She was still comatose at 12.35 p.m. and the respiratory rate had slowed to four per minute. A second intramuscular in-

*From the Children's Hospital, Winnipeg.

jection of picrotoxin (3 mgm.) was given. Within a minute there was a transient generalized convulsion and cessation of respiration. Artificial respiration was done and oxygen was administered, and phenobarbitone (15 mgm.) was given by intramuscular injection. In the following 10 minutes her condition gradually improved, her breathing being stertorous and regular at a rate of 18 per minute. One hour later there was little change except that there were continuous twitchings of her left arm and leg.

At 2.20 p.m. her condition was unchanged. Strychnine (1 mgm.) was given intramuscularly. Ten minutes later there was some response to painful cutaneous stimuli, but twitching of the left arm and leg persisted. At 3.00 p.m. she had a generalized convulsion with cessation of respiration and cyanosis. This responded to suction of the pharynx, artificial respiration and administration of oxygen. Five minutes later 2 c.c. of paraldehyde was administered by intramuscular injection. At 4.00 p.m. her respirations had resumed a normal quiet rhythm and the muscle twitching had ceased. She responded to painful cutaneous stimuli. The knee jerks were brisk and equal on both sides.

At 7.00 p.m. strychnine (1 mgm.) was administered by intramuscular injection, and caffeine sodium benzoate (60 mgm.) by gastric tube. At 7.30 p.m. intravenous therapy was commenced with 5% glucose in water, a total of 400 c.c. being administered over the next 12 hour period. At 9.00 p.m. the infant was awake and talking, but she was drowsy and incoordinate in her movements. The next morning she was still drowsy, but no other abnormality could be made out on examination. She was discharged home and has remained on a maintenance dose of mesantoin (0.045 gm. t.i.d.). On this she has been free of toxic symptoms and of convulsions.

DISCUSSION

Kozol[1] reported the case of a young adult who swallowed 72 tablets (7.2 gm.) of mesantoin in a suicidal attempt. Eight hours later he was found in a deep stupor. His respirations, blood pressure, and pulse rate were within normal limits. He could be roused within 12 hours, and appeared free from ill effects within 36 hours.

Savoy[2] described the case of a 35 year old man who attempted suicide by swallowing 40 tablets of Hydantal (4 gm. of mesantoin and 0.8 gm. of phenobarbitone). After several hours he was found to be drowsy, disorientated, and somewhat agitated. Slight cyanosis of the face was noted. The heart and respirations were normal though slowed down. He recovered after 48 hours.

The present patient became drowsy within 30 minutes and comatose within one and a half hours of swallowing 1.5 gm. of mesantoin. She remained in coma for a period of 10 hours. Her recovery was complete after 24 hours. Coma and respiratory failure were the presenting symptoms. To combat these picrotoxin (3 mgm.) was given by intramuscular injection and repeated after 20 minutes. The second dose of picrotoxin resulted in a generalized convulsion, and thereafter treatment was directed at controlling convulsive episodes. The dose of picrotoxin which was administered was the recommended adult dose (Lucas[3]). To avoid the risk of convulsions a smaller dose would have been advisable in such a young infant. The dramatic improvement in the child after the commencement of intravenous administration of 5% glucose suggested that this procedure may have hastened her recovery.

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MY AMŒBA IS UNAWARE

Of this poem in its favour, though it shares
In my totality. Like adverbs, it qualifies
That to which it is attached, adding
Slowly, carefully, painfully, to my living.
Hosts pay for dinner though the guest
Be uninvited, and symbiosis
Is seldom equal. What most impresses me
Is its immortality, and the "bigness of its littleness."
Truly a marvel of adaptation, equally at home in ponds or paunches
Since the beginning of life, and a threat to religion, with
An ancestry older than all the gods. Not being oviparous,
And multiplying geometrically by diffusion of fission,
Parent and child are the same. Such a conception
Is wholly immaculate, needing no redemption.
Hence no one is born at the expense of another
And death is purely external, an accident
But not a law. Then as its size
Is the reverse of colossal, it seems as far removed
As a prowling space-ship, thus creating
A vastness and mythology in my internal universe
Which makes me macroscopic. Too long
Have the surfaces sufficed us. Beauty, we are mistaught,
Is skin deep, and holy men, lonely in caves,
Have tried to resist temptation by dwelling on the viscera of women,
Thus spitting at heaven and bespattering themselves.
I proclaim equal rights for the parts, the wonder
Of interdependence, the worth
Of the cellular proletariate whose ceaseless labour
Builds the cathedral of eyes and hands. I honour
The encyclopædia of the pseudopodia. The I of the self
Is no less in them than in the entire colony, for individuality
Lies beneath collectivity. But as to a relationship
Unsought by either side, there is need
For bio-justice. None need tolerate
Invasion of frontiers, bacillary insurrections, unicellular anarchy, though such zeal
Be without evil. I am its good, it is not mine, and therein lies
The right of defense. Therefore though I praise
This protozoic ancestor, my pre-Cambrian grandsire,
I aim at its death with all my feeble weapons,
Knowing I do not know if it still survive.

F. R. SCOTT

(This will appear in a forthcoming volume of poems entitled "Events and Signals", the Ryerson Press, Toronto.)

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Clinical and Laboratory Notes

THE OXYTOCIC AND PRESSOR POTENCIES OF CORTICOTROPHIN SOLD IN CANADA*

M. G. ALLMARK and
A. LAVALLEE, *Ottawa*

SINCE THE INTRODUCTION of corticotrophin as a therapeutic agent most manufacturers have been cognizant of the presence of impurities derived from posterior lobe material. During the course of purification some of the activity due to posterior lobe hormones is removed from the product, but there still remains some active material and it is important to know the amount, because an excess of these hormones may produce unfavourable clinical effects.

The present paper is a report on the oxytocic and pressor potencies found in market samples of corticotrophin sold on the Canadian market.

METHOD

The oxytocic potency of corticotrophin products was determined by the method described in the U.S.P. XIV for posterior pituitary extract, and the pressor potency by the rat blood pressure method described by Landgrebe.[1] The reference standard employed for these assays was the Canadian Posterior Pituitary Reference Standard. The time interval between doses for both pressor and oxytocic assays was 15 minutes. For the pressor assays the injections were made into the jugular vein rather than the femoral vein.

RESULTS AND DISCUSSION

In Table I are shown the results of a number of assays on six different brands of corticotrophin for both oxytocic and pressor potencies. Both of the methods used were found to give satisfactory estimates of potency and reproducible results. The usual indications for satisfactory assays were obtained. By repeating the assay two or more times the desired precision may be obtained.

The range of oxytocic units per 25 international units of sample (the usual clinical dose of corticotrophin) was found to be 0.49 to 0.97; and for pressor units 0.43 to 1.09.

Harkness *et al.*[2] do not favour the chicken blood pressure method for determining the oxytocic potency of corticotrophin products because in their studies corticotrophin appeared to reduce the response to subsequent doses of posterior pituitary extract. With an experimental design such as was used for these assays and a 15-minute interval between doses, it has been found that there is very little falling off in sensitivity after the initial dose, for at least seven doses. This permits the completion of one series of doses; for a second series the doses may be raised.

As may be observed from the results shown in Table I the oxytocic and pressor potencies of market samples of corticotrophin examined to

TABLE I.

ASSAYS ON MARKET SAMPLES OF CORTICOTROPHIN FOR OXYTOCIC AND PRESSOR ACTIVITIES*

Brand	*Assay No.*	*Chicken depressor method. Int. units of oxytocic principle*		*Rat pressor method. Int. units of pressor principle.*	
		Per 25 i.u. of sample	*Range in i.u. per 25 i.u. of sample (P.=.05)*	*Per 25 i.u. of sample*	*Range in i.u. per 25 i.u. of sample (P=.05)*
A	1	0.90	0.52 to 1.53	0.79†	0.67 to 0.93
	2	0.90	0.78 to 1.05		
	3	0.60†	0.41 to 0.89		
B	1	0.97	0.72 to 1.31	0.94	0.74 to 1.17
	2	0.97	0.84 to 1.12	1.00	0.87 to 1.14
C	1	0.89	0.76 to 1.04	1.09	1.00 to 1.17
	2	0.89	0.83 to 0.95	0.83	0.43 to 1.60
	3	0.96	0.77 to 1.20	0.91	0.67 to 1.22
D	1	0.92	0.82 to 1.03	0.43	0.37 to 0.50
	2	0.70	0.48 to 1.01		
E	1	0.49	0.44 to 0.54	0.47	0.42 to 0.53
F	1	0.77	0.45 to 1.31		

*Canadian Posterior Pituitary Reference Std. No. H-69605 used for these assays.
†Weighted result of two or more assays.

*From the Pharmacology and Toxicology Section, Food and Drug Laboratories, Department of National Health and Welfare, Ottawa, Canada.

date are all quite low. From a clinical standpoint it is not considered that an amount of oxytocic and pressor principles less than one international unit per dose of corticotrophin would have any noticeable effect on the patient.

SUMMARY

A survey of the pressor and oxytocic potencies of different brands of corticotrophin sold on the Canadian market is presented. The pressor potency ranged from 0.43 to 1.09 units; and the oxytocic potency from 0.49 to 0.97 unit per 25 units of corticotrophin.

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CORONARY ARTERY INSUFFICIENCY WITH LEFT VENTRICULAR ENLARGEMENT AND FAILURE TREATED BY EPICARDECTOMY AND MEDIASTINAL CARDIO-OMENTOPEXY*

PRELIMINARY REPORT

ARTHUR VINEBERG, M.D., *Montreal*

IN PREVIOUS PUBLICATIONS in this Journal[1 to 4] we have presented evidence, both experimental and clinical, to indicate that coronary artery insufficiency can be successfully treated by implantation of the left internal mammary artery into the left ventricle. In the past year this procedure has been supplemented by applying to the surface of the heart two pericardial fat grafts at the same time as implantation of the internal mammary artery. Twenty human cases have now been treated by the double procedure with what appears to be a high percentage of good results. Recent experimental data, still unpublished, indicate that the application of pericardial fat grafts to the heart results in the development of anastomoses between the blood vessels in the heart and those in the pericardial fat grafts.

Unfortunately, the implant procedure cannot be performed on patients with a markedly enlarged heart, or upon patients in left ventricular failure—the operative risk is too great. We have seen many such patients and have been forced to send them home without surgical treatment; quite a few of them have died a few months later.

As a result of this experience we have sought for a simple means of bringing fresh blood to a failing, enlarged heart.

Omental graft to the heart.—O'Shaughnessy[5, 6] first reported the use of the omentum as a source of revascularization of the left ventricle. His experimental work was very convincing and was done upon whippet dogs; he performed the operation upon human cases, but the results of his human cases have not been reported because of his untimely death on the beach at Dunkirk.

There were three distinct disadvantages to the procedure as done by O'Shaughnessy: (1) The abdominal-thoracic approach was used, thus making it unsuitable for cases with left ventricular failure. (2) The left leaf of the diaphragm was split to bring the omentum into the chest; this resulted in thoracic herniation and other unpleasant complications. (3) O'Shaughnessy was unaware of the advantage to be gained by removing the epicardium before applying the omental graft.

MEDIASTINAL CARDIO-OMENTOPEXY

We wish to report a modification of the O'Shaughnessy operation which is simple and which does away with the objections above listed. In this procedure, a U-shaped incision is made from the left 6th rib to the midline anteriorly, extending into the epigastrium inferiorly in the midline. The peritoneal cavity is opened, and the omentum identified. The attachments of the peritoneum to the xiphoid cartilage are cut; this permits the index finger to be inserted directly into the anterior mediastinal space anterior to the heart so that the left pleura can be pushed laterally. Removal of the 6th costal cartilage exposes the pericardium directly. The pericardium is opened, the epicardium removed, the opening from the abdomen to the mediastinum is enlarged by cutting the attachments of the diaphragm to the xiphoid cartilage, and the omentum is then pulled through this archway so as to lie directly on and around the entire left ventricle, where it is fixed. By this procedure the omentum is placed directly on the heart without going through the leaf of the diaphragm, thus obviating herniation, diaphragmatic pull, and constriction of blood vessels in the pedicle.

This operation was performed on July 20, 1954, on Mr. H.W., inspector, a 52 year old male, at the Jewish General Hospital, Montreal. In September 1952, he had developed anginal pain of short duration while walking. A few weeks later he had a severe attack of substernal pain radiating down both arms and requiring hospitalization. In spite of 12 months' treatment with dicoumarol his anginal pain persisted on slight effort. A second attack of coronary thrombosis occurred in October 1953, with four weeks' hospitalization. After this

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attack, his exercise tolerance was limited to about 30 yards. His third attack of coronary thrombosis occurred in May 1954, six weeks prior to admission, leaving him totally incapacitated. On admission, he was suffering anginal pain on the slightest effort or excitement. This was relieved by nitroglycerin. There was pain after eating and swelling of the ankles, with moderate orthopnœa. He required three pillows to sleep, otherwise experienced a choking sensation when lying flat.

Examination.—Adult male, cyanotic tinge to lips and finger nail beds, with some shortness of breath at rest. B.P. 230/140. P.M.I. in A.A.L., L.B.C.D. in A.A.L. Peripheral arteries thickened and tortuous. Liver edge 2 f.b. below rt. costal margin, moderately tender. Spleen palpable laterally 1 f.b. below lt. costal margin. ECG showed previous large antero-septal infarction, and left heart strain pattern. Chest radiograph showed enlarged left ventricle with marked tortuosity of thoracic aorta. Renal function showed some impairment, although Mosenthal test showed concentrations from 1010 to 1020, with a night output of 320 c.c. Albumin ++. NPN on first examination 44.1 mgm. % settled to 35 mgm. %. Sedimentation rate 45 mm.

The exercise tolerance on July 7, 1954, was 6 steps before substernal pain developed. The patient was digitalized and given mercuhydrin. By July 12, 1954, he was capable of doing 15 steps and, on July 16, 23 steps.

On July 20, 1954, a U-shaped incision over the 6th left rib and down the midline to the epigastrium was made. The abdomen was opened and a large vascular omentum easily mobilized. The mediastinum was entered beneath the xiphoid cartilage, and the left pleura pushed to one side. The left costal cartilage was removed and the pericardium opened. In addition, removal of the 6th rib was necessary in order to expose the left ventricle which was out in the axilla. Part of the epicardium was removed and the omentum was drawn through the anterior mediastinal space, where it was fixed to the left ventricle and fibrous epicardium.

The patient's postoperative course was uneventful. He sat up in bed the night of operation, on the side of the bed three days later, and was discharged from hospital on the 11th postoperative day.

It is too early to know the results of this procedure. However, a large vascular body has been attached to an enlarged hypertensive heart in failure. The omentum is placed on the heart through the anterior mediastinum, doing away with the necessity of bringing it through the diaphragm with the known complications.

The operation is done with the patient lying on the back, resulting, in this particular patient, in no disturbance whatever, as evidenced by the absence of changes in pulse and blood pressure.

The co-operation of Drs. Harold and Sydney Segall in the preoperative and postoperative treatment of this patient is gratefully acknowledged.

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PROTECTION FROM SCATTERED RADIATION DURING FLUOROSCOPY

S. J. SHANE, M.D.* *Sydney, N.S.*

IN THE MODERN tuberculosis hospital a fluoroscopic examination is usually carried out before and after each pneumothorax or pneumoperitoneum refill. Under average circumstances, a physician in such a hospital usually gives about 10 or 15 pneumothorax or pneumoperitoneum refills during each working morning, thus undergoing 20 or 30 exposures to scattered radiation each day. Though each exposure may last only a few seconds, the sum total of such daily radiation, spread over a period of years, may add up to a considerable quantity. The usual method of protecting oneself against such exposure is to don a lead-rubber apron and gloves before each fluoroscopic examination. It is obvious that, under the circumstances cited above, it might be necessary to put on and take off this heavy protective equipment a total of 20 or 30 times during a pneumothorax or pneumoperitoneum clinic. This being the case, one might expect a degree of laxity among resident physicians, in providing themselves with protection under such circumstances; and, in our own experience, this has been the case.

In some tuberculosis institutions, partial screening is provided by suspending a rather narrow strip of lead-rubber from the fluoroscopic screen. We believe that the protection provided by this arrangement is practically negligible; and in any case it is still necessary to put on and take off the protective gloves, at least 20 or 30 times during the average refill clinic.

It therefore seemed desirable to construct a protective device that would afford a similar degree of screening to that with an apron and gloves, but which would, at the same time, be so simple to manage that there would be little temptation to disregard the protection it could offer.

Such a device was constructed at this hospital, and is illustrated in the accompanying photographs.

In essence this protective device consists of a rigid arm of metal "angle-iron," measuring approximately four and one-half feet long. This is welded to two pieces of bar-iron, in such a fashion as to form a right-angled triangle. This triangle is attached to two solid metal brackets bolted to the wall, in such a manner that the apex and the right angle are hinged and the third angle is free. This method of hingeing makes it possible for the entire triangle to fold against the wall when not in use (Fig. 1), and the two pieces of bar-iron add further rigidity

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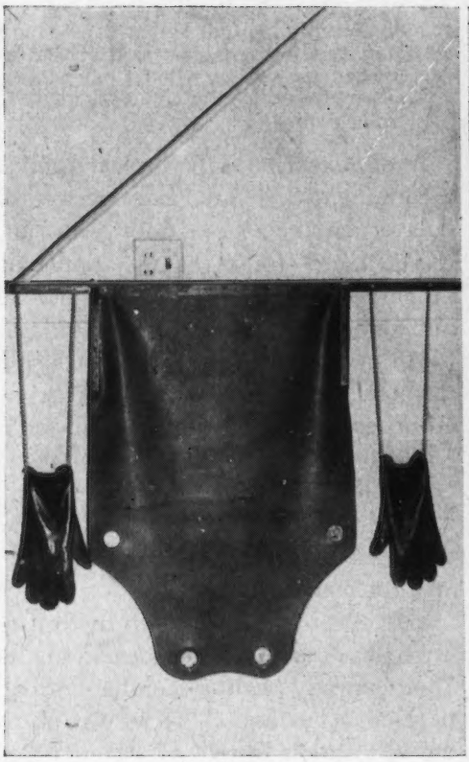

Fig. 1

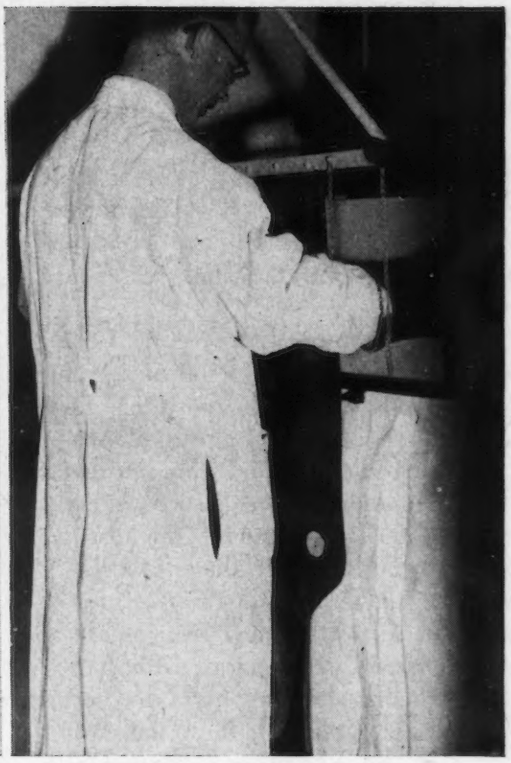

Fig. 2

to the cross-bar. A lead-rubber apron is then riveted upside-down to the cross-bar, in such a position that, when the triangle is swung forward, the apron hangs directly in front of the fluoroscopic screen. The height at which the triangle is bolted to the wall, is such that for a physician of average stature, the apron protects him from just below the shoulders to well below the knees. A pair of fine but strong chains is then riveted to each glove, and then to the cross-bar on each side of the apron (Fig. 1). These chains are of such length that one's hands can be thrust into the gloves without difficulty, the gloves are hung palms outward with the thumbs directed medially.

When one is prepared to carry out a fluoroscopic examination, it is only necessary to swing the triangle forward in front of the fluoroscopic screen, insert one's hands into the gloves, and proceed with the examination (Fig. 2). The gloves are of such a weight that they will fall from the fluoroscopist's hands when he raises them after completing the examination, and it is unnecessary to use the other hand to remove the glove from either hand.

Having used this device over a period of almost two years, we have found that it has certain advantages: (1) It apparently provides the same amount of protection to the fluoroscopist as provided by the apron and gloves used in the conventional manner. (2) No time is wasted by the operator in donning and removing apron and gloves during a refill clinic. (3) It is so simple and convenient that there is no temptation to disregard the protection that it provides.

It is possible that radiologists, other than those working in tuberculosis hospitals, may wish to avail themselves of the protection provided by this device.

THE CUTANEOUS APPLICATION OF A NICOTINIC ACID CREAM AS A DIAGNOSTIC AID IN VARIOUS RHEUMATIC DISEASES*

de GUISE VAILLANCOURT, M.D.,
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RECOGNITION of typical cases of rheumatoid arthritis presents little difficulty. In the early stages of the disease, however, or when the process is atypical the diagnosis can often be extremely difficult to establish and the clinician

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has to be satisfied with a probable or tentative diagnosis. Not infrequently, a six-month observation period is necessary before rheumatoid arthritis can be identified with some degree of certainty.

Although the diagnosis of rheumatoid arthritis rests primarily on an adequate evaluation of symptoms and signs, radiography and laboratory procedures are helpful in establishing the nature of the disease.

The erythrocyte sedimentation rate and a complete blood count are probably the most valuable laboratory aids in the diagnosis of rheumatoid arthritis. Two other procedures, the streptococcus agglutination test and the sensitized sheep cell agglutination test are also considered helpful in some 60% of cases of the disease. The combined results of all these laboratory procedures more than the evaluation of any of these tests taken separately may help in establishing the rheumatoid nature of an arthropathy.

In an effort directed at finding more diagnostic aids, we studied the application of an interesting observation made by Nassim and Banner[2] and also by Oka.[3] These investigators reported in patients suffering from rheumatoid arthritis the absence of the usual local hyperæmic reaction observed following the topical application of a 5% water-miscible cream containing the tetrahydrofurfuryl ester of nicotinic acid.* A similar phenomenon was reported by Streitfeld and Saslaw[4] in a small series of patients suffering from rheumatic fever. The above preparation has been used in Europe[1] and more recently in our clinic as an effective rubefacient.

Materials and methods.—To perform the test, the examiner gently rubs with his finger 80 mgm. of the nicotinic acid cream into a cutaneous zone of approximately one and a half inches diameter on the volar aspect of the left forearm. A lanolin-vaseline ointment used as an inert vehicle for the nicotinic acid preparation† was applied in the same manner on the corresponding area of the right arm and was used for control purposes. In all instances, the test and control areas were observed for hyperæmia and œdema by the same investigator, who was completely unaware of the probable diagnosis.

The skin response to the test cream was thus graded:

−: No visible alteration of the skin or very slight erythema fading out within five to ten minutes.
±: Questionable hyperæmia and/or local œdema.
+: Hyperæmia with or without œdema, associated with a sensation of warmth and tingling at the site of application.

In our experience the topical application of the nicotinic acid cream may occasionally induce delayed cutaneous reactions occurring from four to 12 hours following the application of the preparation. Since all those who reacted to the application of the test cream did so within 10 to 15 minutes, we decided to consider negative the erythematous reactions occurring following an observation period of at least one hour.

We observed that the normal response to the cutaneous application of the nicotinic acid cream is more rapid and more intense if the subject is young, if the skin is warm and if the person is of light complexion.

Skin testing with the tetrahydrofurfuryl ester of nicotinic acid was performed on 163 subjects; 72 apparently healthy members of both our resident and nursing staffs served as controls. Of the 91 patients tested, 57 suffered from rheumatoid arthritis, five from rheumatoid spondylitis, four from rheumatic fever, two from disseminated lupus erythematosus, 10 from osteoarthritis, and 13 from undiagnosed arthropathies.

TABLE I.

SKIN REACTIONS TO THE TOPICAL APPLICATION OF THE TETRAHYDROFURFURYL ESTER OF NICOTINIC ACID IN RHEUMATIC DISEASES AND IN CONTROLS.

Diseases	*Number of cases*	*Responses* +	±	—
Rheumatoid arthritis	57	4*	2	51
Rheumatoid spondylitis	5	3	0	2
Rheumatic fever	4	0	0	4
Undiagnosed arthropathies	13	10	0	3
Disseminated lupus erythematosus	2	1*	0	1
Osteoarthritis	10	10	0	0
Controls	72	69	2	1
Total	163			

*Patients treated with either cortisone or corticotropin

Results.—In all instances the application of the control ointment to the right forearm failed to induce any cutaneous reaction. Our results are otherwise tabulated in the accompanying table.

All the controls but three presented within five to 10 minutes local hyperæmic reactions with or without œdema after cutaneous application of the nicotinic acid cream. One member of this group failed to react although some tingling and

*Manufactured by Ciba Co. Ltd. (Montreal) under the trade name "Trafuril" and generously supplied through the courtesy of Dr. Fred Wrigley, Medical Director.
†Ciba's "Ointment 'B'".

warmth but questionable hyperæmia were observed. The 10 patients suffering from osteoarthritis reacted like the controls to the cutaneous application of the cream.

Of the 57 rheumatoid arthritis patients, only six responded to the local application of the nicotinic acid cream. Four patients from this group were receiving either cortisone or corticotropin at the time of testing. The two other patients were treated with phenylbutazone.* In rheumatoid spondylitis the results were much less predictable: we observed three positive and two negative reactions.

Only four patients suffering from active rheumatic fever were skin-tested; all of them failed to react to the topical application of the nicotinic acid cream. This is in agreement with the observation previously mentioned.[4]

Of the two disseminated lupus erythematosus patients tested, one did not respond but the other, receiving huge doses of cortisone, reacted normally to the skin test.

Finally, in three of 13 patients suffering from undiagnosed arthropathies, the nicotinic acid cream did not induce a local hyperæmia. Considering the frequent atypical manifestations of rheumatoid arthritis, such a diagnosis in the nonreactors of this group remains a distinct possibility.

DISCUSSION

We are not prepared to offer any explanation concerning the mechanism of the phenomenon observed. It is however interesting to note that cortisone, corticotropin and possibly phenylbutazone seem capable of modifying the reaction.

The present series is obviously too small to justify any conclusions regarding the value of the skin test in rheumatic diseases. Our data as well as reports from elsewhere[2, 3, 4] suggest that patients suffering from certain rheumatic diseases might react differently to the cutaneous application of the tetrahydrofurfuryl ester of nicotinic acid according to the variety of arthropathy involved. Since this chemical compound seems more or less inert in patients with active rheumatic fever or rheumatoid arthritis and almost consistently induces a strong hyperæmic response in normal individuals and in certain arthropathies, the nicotinic acid cream might prove to be helpful as a diagnostic aid in rheumatic diseases.

*"Butazolidin"—Geigy Pharmaceuticals, Montreal.

SUMMARY

The cutaneous application of a cream containing 5% of the tetrahydrofurfuryl ester of nicotinic acid to 163 subjects induced a marked localized hyperæmic reaction in all but one apparently healthy individual serving as controls, and in all patients suffering from osteoarthritis. In those with active rheumatoid arthritis and in rheumatic fever, the drug failed to produce any cutaneous reactions except when either cortisone or corticotropin was being therapeutically administered; two rheumatoid arthritis patients receiving phenylbutazone also failed to respond to the cream. The reaction appeared unreliable in rheumatoid spondylitis. One patient suffering from disseminated lupus erythematosus and receiving cortisone responded normally to the local application of the cream; an untreated case of the same disease did not react when skin-tested.

The mechanism of this phenomenon and further observations regarding the value of the nicotinic acid cream as a diagnostic agent in rheumatic diseases are being investigated.

The helpful assistance of Miss Jeannine Leblanc, R.N. is gratefully acknowledged.

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ETHYLENE GLYCOL POISONING

In contrast to the two cases of ethylene glycol poisoning in children recently reported in this journal (Nadeau, G., Cote, R. and Delaney, F. J., *Canad. M. A. J.*, 70: 69, 1954), four cases reported from Northern Italy all occurred in persons who should have reached years of discretion. The four men concerned, aged from 56 to 70 and described as heavy eaters and drinkers, were ill advised enough to take from 100 to 1,000 c.c. of ethylene glycol as a beverage. The results were manifested in three stages: an initial one of drunkenness and euphoria; a second of confusion, inability to walk and vesical sphincter disturbances; and a third of deepening coma from which three men never recovered. The final stage was accompanied by signs of severe renal damage, with oxalates and erythrocytes in the urine.

It would seem that the three stages may correspond to one of alcoholic intoxication, one of mixed alcoholic and oxalic intoxication, and one of oxalic acid poisoning alone. The severity of the condition is directly proportional to the quantity drunk, with a minimum lethal dose of over 100 c.c.

Chemically, the likelihood is that the syndrome will be confused with ethyl alcohol intoxication, but the whole picture is more severe and of more rapid development in ethylene glycol poisoning.—I. Morini, *Minerva Med.*, 45: 72, 1954.

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Editorials

PSYCHOTHERAPY IN PRISONS

In this century there has been a continued enlightenment as regards prison environment, and the after care of the ex-prisoner has made some advances. One of the greatest advances has been the provision of psychotherapy in jails throughout the world. The desirability of more scientific treatment and the lessening of daily deprivation have come more to the forefront. Mackwood[1] has recently discussed some of the difficulties encountered in the application of psychotherapy in a prison. It is necessary for the custodial staff to have special training to understand that psychotherapy may act through releasing instinctual energy in psychoneurotic delinquents; these patients may need to "work through" their uncovered aggressive urges, which involve behaviour which is punishable in prison. A staff not psychiatrically trained is uneven and does not understand the factors involved; special centres are necessary, because control cannot be expected in an ordinary prison where many different types of offender are kept under the same disciplinary regimen. Such special centres where the offenders are kept separately from the other prison inmates protects them from the unfavourable attention of their fellows not receiving such treatment; one great advantage is that group therapy can be started.

The effect of imprisonment is to take the person back to childhood conditions, where he is under authoritarian control. Roper[2] comments that such behavioural regression may be an asset, imprisonment recreating in some measure the former family and playgroup situation and so allow the "working over of the actualities of the early formative period." The same author, in commenting on the difficulties of psychotherapy in prison, points out that a number of factors are involved. Some difficulties are associated with selection of the patients to be treated, and the timing of such treatment. Other difficulties are associated with after care or the time of the sentence; this may be too short, resulting in interruption of treatment, or too long, producing despair. Relatives can be a help or a hindrance; in the latter case by fostering ideas of injustice or innocence. Domestic difficulties are one of the greatest bars to progress, but of course these may also be insoluble by psychotherapy outside the prison walls. Difficulties associated with the prisoner group are very important. Prisoners as a whole tend to have a low opinion of psychiatric patients, especially those who are sexual offenders: this may result in reluctance to accept therapy. Dr. Roper gives some encouraging figures of the results of psychological treatment, although it is early to draw definite conclusions; out of 108 treated cases who have been at liberty for varying periods since the project started in 1947 only six have been traced back to prison again.

We must hope that continued enlightened treatment of offenders will reap rewards, and that a better social conscience will eventually result.

W.F.T.T.

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VASCULAR HEADACHE

["In the headache he orders the opening of the vein of the forehead." Arbuthnot (Quoted by Samuel Johnson.)]

Wolff[1] had shown that the headache of migraine is due to vasodilatation of the extracranial vessels. Recently Wolff and Tunis[2] have investigated the results of taking artery pulse wave contour records two to five times weekly on 40 patients with vascular headaches of the migraine type. These recordings were taken both during and between the headaches, and also on normal subjects who rarely had headaches. The authors comment that greater variation in the contractile state of the vessels is found in migraine subjects compared with the normal, even in headache

free periods: and that 72 hours prior to a headache the variability is increased. Of interest is the fact that 24 hours prior to the onset of a headache there is increased vasoconstriction, which becomes maximal a few minutes before the headache starts.

During the headache itself distension, dilatation and intramural œdema occur. Wolff *et al.*[3] have shown that, elsewhere in the vascular bed, for example the conjunctivæ, vasodilatation occurs on the side of the lesion but that the blood flow rate was reduced as much as fivefold compared with the contralateral side. More interesting still is the presence of hæmatoma around the vessel, which the authors have described as occurring up to 6 cm. in diameter and 2 cm. in depth: these hæmatomas were usually localized near the frontal branch of the temporal artery which was involved in the dilatation. It is well known, of course, that dilatation is not the entire answer to the question of headache; as Kunkle and Wolff[6] point out there is considerable dilatation of extracranial vessels after vigorous exercise but this is not painful.

Wolff *et al.*,[3] using apparatus to assay deep pain and superficial pricking pain thresholds, have shown that lowered deep pain threshold about the extracranial vessels and the surrounding tissues was an invariable accompaniment of the headache. The authors comment that vasodilatation was an essential requirement, but that lower threshold alone or moderate vasodilatation by itself did not cause headache. This was shown by the fact that dilatation of extracranial vessels following ultraviolet irradiation or injection of sodium chloride solution into the subcutaneous tissues of the head did not produce a headache.

Peters[4] states that migraine is a psychosomatic disorder with a definite hereditary tendency which occurs in over-conscientious and perfectionistic individuals; but in the same symposium Giffin[5] points out that, although the migraine diathesis may be related to genetic factors, the fact that a parent and children have migraine may be correlated with psychiatric relationships. Giffin comments on the migraine personality with its strong perfectionistic traits, which are often developed to protect against insecurity and as means of expressing anger. Psychiatric help may be indicated in some migrainous patients; but we must not forget that the psychological factors in headache are not a recent innovation, Plato some two centuries B.C. stated: "Let no one . . . persuade to cure the head until he has first given you his soul to be cured."[6]

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Editorial Comments

TETANUS IMMUNIZATION

Tetanus is one of the diseases which can and ought to be abolished. It is well-known that, thanks to active immunization of servicemen, the U.S. Army Medical Services had to deal with only 12 cases of tetanus out of over 2,700,000 hospital admissions for wounds and injuries in World War II. Of these men only four had been properly immunized and of the latter only one died.

The questions now arise: (1) whether immunity after a properly carried out basic immunization course lasts for an appreciable number of years; (2) whether routine prophylaxis after injury, even years after immunization, may not take the form of a booster dose of tetanus toxoid rather than the injection of tetanus antitoxin with its attendant danger of anaphylactoid reaction.

The results of a study carried out by members of the staff of Johns Hopkins Hospital (*Bull. Johns Hopkins Hosp.* **94**: 204, 1954) suggest answers to these questions. Serum antitoxin levels were determined on 145 adults previously immunized with toxoid. Of 74 whose immunization had taken place between five and 11 years previously, only six had less than 0.01 unit antitoxin per ml., and 45 had over 0.1 unit per ml., a level generally assumed to be protective. Of 73 who had had a booster dose within five years, all but nine reached the 0.1 unit level, and 45 had more than 1.0 unit per ml. Furthermore, giving a 0.5 ml. booster dose of tetanus toxoid to persons in the former group led in all of 54 tested to a 10-fold to 1,000-fold rise in antitoxin titre by the seventh day.

It would seem, therefore, that practitioners could maintain a satisfactory level of immunity in veterans by giving a booster dose of tetanus toxoid at 5-year intervals, or even, in the majority, at 10-year intervals. It is also recommended that such persons treated for injuries in civilian practice should receive tetanus toxoid alone as prophylaxis, and that as part of a campaign to increase the incidence of immune

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members in the population anyone given antitoxin should simultaneously receive toxoid at a different site.

UNDERGRADUATE EDUCATION

Efforts to adapt the curriculum of medical students to present-day needs continue. The U.K. College of General Practitioners, mindful of the need for adequate preparation of a fair proportion of United Kingdom undergraduates to play the exacting part of family doctors, has set up an Undergraduate Education Committee. This Committee now reports (*Brit. M. J.*, 1: 1146, 1954) an analysis of a questionary addressed to some of the recently formed regional faculties of the College, in order to find out what opinions were held by general practitioners themselves on medical training. This analysis deserves attention, since the keen general practitioner knows better than anyone what parts of his undergraduate training were superfluous and what essential aspects of his education were neglected.

The tendency to make the student specialize in scientific subjects in his last one or two years at school is deplored by the faculties; it is suggested that the student's time at this period should be evenly divided between a general education and an approach to scientific subjects.

In student selection for medical schools, it is felt that character, home background, heredity and school record should rank as high as mere academic achievement in scientific subjects; it also seems logical to have an experienced general practitioner on selection boards to assist the dean.

As is customary in such reports nowadays, the length of time devoted to instruction in anatomy and advanced surgery comes in for criticism, and teachers are also attacked for demonstrating obscure conditions in preference to simple and common variations in health and disease.

The need for giving the student more insight into general practice is stressed. Examples of techniques for introducing the student to the management of the patient outside hospital are given. Thus, one London consultant takes a student with him to his domiciliary visits; the use of general practice teaching units (as at Edinburgh) or of attachment of students to general practitioners is also mentioned. In any case, opportunities must be made for the student to come in contact with general practitioners right through his course, so that he may appreciate the great volume and variety of service which a general practitioner has to render.

Anyone who has been flung upon the uncharted sea of general practice without the navigational aid of a family tradition in the art must fervently acclaim the suggestions of the College, and hope that they will bring forth fruit.

Men and Books

CHOLERA IN QUEBEC IN 1849

SYLVIO LEBLOND, M.D.,* *Chicoutimi, Que.*

CHOLERA HAS APPEARED several times in Canada. Some of the epidemics were brought into the country at Quebec, which was then the sole port of entry for ships from Europe. In 1832 cholera appeared in Quebec, the infection having come from Dublin on the *Carrick*. It started in rue Champlain, like all the other epidemics, and carried off 3,451 persons out of a total urban population of not more than 30,000.

In 1834, there was another cholera epidemic. The 1832 outbreak had led to a government decision to construct at Quebec a hospital for seafarers and immigrants, designed for the treatment and care of sailors and immigrants who otherwise had no lodging but the rooming houses in the Quartier Champlain, where they were poorly installed and particularly badly cared for when ill.

Although the hospital was unfinished, it opened its doors in 1834 for the reception of cholera patients, only to close again in October when the epidemic was at an end. Construction continued and it was officially opened in May 1835. During 1834 cholera had claimed 2,509 victims in Quebec. It reappeared in 1849 and this time, 1,185 inhabitants of Quebec died.

The 1849 epidemic is of interest because of the development of medical ideas at this time, the statement being made in some quarters that the disease was contagious, a fact hitherto denied. It was believed that smallpox, typhus and typhoid were contagious diseases transmitted by contact with individuals or fomites, but this mode of transmission was not considered applicable to cholera, which appeared only as a result of unknown changes in the atmosphere. Some went so far as to attribute cholera to an atmospheric surplus of ozone, and used this theory to explain on chemical grounds the good effects of sulphur and wood charcoal on the disease.

In 1849 cholera came from the United States via Kingston, appearing relatively late in Grosse Isle (quarantine) at a time when the epidemic was on the wane in Quebec itself. Quebec was the port of entry for all ships coming from Europe, and every ship had to submit to quarantine inspection at Grosse Isle during the navigation season. The Inspector had orders to use firearms if necessary to exact obedience from a recalcitrant ship's captain. The volume of emigration from the British Isles was very great, particularly from Ireland where famine was

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widespread. In 1848, Europe had been invaded by cholera, and in 1849 it was still rampant in England and especially in Ireland. It was hoped that it would not cross the sea to Quebec.

In December 1848, Dr. Painchaud had given a public lecture on "Asiatic cholera," ending with the statement:

> "If cholera is in England, it will have plenty of time to burn itself out before the merchantmen leave for Canada. Even if there were cholera in New York, it could not cross the border during winter; it might well ravage all the Southern States, but it would be gone before the snows melt. However, its appearance is possible, and it is up to us to be on our guard." (*Journal de Québec*, January 4, 1849).

Conditions of hygiene in Quebec were not calculated to favour prevention of the appearance or spread of the disease, for there was neither a piped water supply nor a sewerage system. Water was obtained from the numerous springs, from wells or even from the St. Lawrence, either at the mouth of the St. Charles river or at the quay-side. There was an organization of water carriers distributing water to a population of about 37,000. Sewage and refuse accumulated in courtyards, stables and streets, giving rise in certain quarters to almost suffocating smells. Some householders had installed private systems carrying off wastes to the river, but these discharged their contents at the very spots where drinking water was obtained for the population.

It was the 1849 cholera epidemic which led to a resolution by the Quebec Board of Health stressing the urgency of installing in the town, whose rocky foundation was ill adapted to absorption, an aqueduct for household water supply, as well as sewers. Water was obtained from the Indian reserve at Lorretteville, and the aqueduct began to function in 1854. This was a great improvement, but sewers were not installed until somewhat later.

Cholera appeared on July 4 in the Champlain district of the Lower Town of Quebec. This district was situated at the foot of the cape, between the latter and the St. Lawrence. It consisted only of a single, long, narrow street built up on both sides. A poor and numerous population lived there all the year round, augmented during the navigation season by a tightly packed collection of sailors and immigrants. At the end of the street, at what was known as the cul-de-sac of the Lower Town, drinking water was obtained. The filthy state of this place was sufficient, according to the partisans of the non-contagion theory, to pollute the atmosphere and favour the development of cholera.

On the other hand, the champions of contagion attributed the outbreak to the presence of mattresses thrown overboard from sailing ships on which cholera had been present and drifted to the cul-de-sac of the Lower Town where the drinking water was obtained.

The epidemic spread very rapidly through the district and the town. A shoemaker called McGill was the first victim. He died within a few hours on July 4.

A Central Board of Health had been formed at Montreal by Act of Parliament (Act 12 Vict. ch. 8). It was composed of Drs. Wilfred Nelson, Guillaume Deschambault and Robert Ley MacDonnell and of Messrs. Olivier Berthelot, William Workman, John James Day and Moses J. Hayes. Dr. Aaron H. David was secretary. It had drafted orders and regulations, with special reference to cleanliness and cleansing of streets, courtyards and outhouses. All local Boards were obliged to furnish it with reports.

At Quebec, a Board of Health had been in existence since 1832, and took up its functions each year as required. It had been very active in 1832, 1834 and 1847. It had acquired considerable experience, since the same membership had been retained so far as possible. In this year (1849) the nominees were Messrs. Légaré and Sewell, Drs. Morrin and Nault, Mr. Boxer, Drs. Paradis, Joseph Painchaud and James Douglas. Most of these had sat on the Board during the 1847 typhus epidemic. Mr. Robert Symes was appointed sanitary inspector at a salary of ten shillings a day. F. X. Garneau, who was then preparing the History of Canada which was to render him famous, was the secretary and Dr. Morrin the chairman.

Dr. Joseph Morrin was a very busy man with a well developed social sense. He had just founded a medical school at Quebec. With Dr. J. Douglas, he did much work for the Beauport Asylum, and was an alderman of the city of Quebec. Laval University changed his medical school into a Faculty of Medicine, and he left it a sufficient sum in his will to establish a prize still known as the Morrin Prize.

The Board of Health was very active. It organized domiciliary visits, and four doctors (Bardy, Wolf, Robitaille and Carrier) were designated for these visits, with the duty to report every case of illness, cholera or otherwise. These reports, evidence of a well-developed social sense, contributed largely to the cleansing of the town, its streets and the quays of the old City, to the movements for installation of a water supply and sewerage systems, and even to the improvement of the city finances.

CONTAGION OR NON-CONTAGION

The general opinion at this time was that cholera was not a contagious disease in the same sense as smallpox, typhoid or typhus. Contact with or proximity to cholera patients did not lead to cholera. As examples, the doctors, nurses, priests and relations who came in close contact with patients without harm were cited, in contrast to typhus which, two years earlier, had taken a heavy toll of the doctors, priests and hospital personnel, particularly on Grosse Isle. How-

ever, from time to time a warning voice was raised.

In July 1849, an alarmist who signed himself "Delta" had written the following in the *Gazette* and *Mercury*.

"I am an alarmist and I want to do what the Board of Health won't do—alarm the public, so that it won't run unnecessary risks. Fear does not cause cholera but fear can predispose to it; if in such a state you come in contact with the disease you may contract it and probably will."

Poor "Delta" was attacked by the medical profession, the citizens and the newspapers, and one fine day he decided to break off the argument. The *Quebec Mercury* published (August 28, 1849) a humorous account of his journalistic funeral. He had been accused of frightening a certain number of citizens who had in consequence taken the cholera and died.

Against the contagion theory, the argument was used that individuals in perfect health and with absolutely no contact with cholera patients were suddenly taken with cramps, profuse diarrhœa, and coldness and were carried off in a few hours, while others who carried the corpses resisted infection entirely; hospitals containing cholera patients in thickly populated parts of London and elsewhere had not conveyed the disease, and few cases of cholera had been seen among the medical and other personnel.

Dr. John Hall, a Quebec surgeon, published a long article in the *Quebec Mercury* on August 18, against the contagiousness of cholera, ending by quoting the conclusions of the Board of Health of New York City, which had appeared in the *New York American Journal* in July of that year.

"The undersigned believe that the cause of the disease exists in the atmosphere and that the whole of the community are at present more or less under the influence of the peculiar condition of the atmosphere . . . and in this way predisposed to the disease. To develop the disease, however, existing causes are necessary, and these are to be found in all those things which have a tendency to disorder the bowels. With regard to the peculiar condition of the atmosphere which predisposes to the disease, we know nothing. Human skill and agency, therefore, can do nothing in meeting this difficulty. The existing causes, on the contrary are in a great measure under our control, and by properly guarding against these, much, very much may be done in obviating the development and extension of the disease." Signed: John Beck, M.D., Sam'l W. Moore, M. David, Joseph Smith."

Hence it was generally believed that the disease was in the atmosphere and that it developed only in the presence of certain favouring factors in the individual, in his behaviour or in his atmospheric environment. Dirtiness, mephitic and nauseating odours, fatigue, anxiety, fear, lack of food, and especially the use of alcohol were certain to lead to the appearance of the disease.

There were sceptics, however, who thought that all this campaign for street and court cleansing was fit only to scare the populace. The *Quebec Morning Chronicle* of July 23 said:

"With all respect for our contemporaries . . . we state plainly, dogmatically and confidently, that neither diet nor cleanliness have anything to do with the matter; whether chloride of lime, of zinc, burning barrels of tar or draining cesspools are vain and useless in the open air. The drains and sewers smell no worse this year than last, vegetable matter decays not more speedily this summer than it did in July 1848, the poor are not dirtier, nor the rich cleaner than they ever have been; poverty and sloth produce dirt now as ever, and wealth revels on clean linen and drops asleep on comfortable beds and well aired bed rooms at present as they have been wont; yet both classes are seized with cholera, and the disease shows little favour. . . ."

Dr. von Iffland, a distinguished Quebec physician then attached to the Beauport Asylum which Dr. James Douglas had founded in 1845, sent a long letter to the *Canadien de Québec* on the non-contagiousness of cholera. The letter was published on June 18, some weeks before the disease appeared. According to him, cholera was not contagious or else the human race would have ceased to exist. In contrast to smallpox, this disease could attack the same person several times; if it were contagious, it would disappear only when there were no victims. He explained that all epidemic diseases had their origin in the same thing, the state of the air. This was the best solution that could be given, although the actual atmospheric conditions favouring or producing these diseases were unknown. It was a fact that these epidemics appeared particularly when the atmosphere was moist and hot. Von Iffland added, "The atmospheric changes at these times will presumably always remain unknown."

Like many others, Dr. von Iffland did not know that, in Paris, Pasteur was already finding a reply to these questions and that the reply would destroy all these fine theories. Nor could he foresee that in 1885 Koch would demonstrate the cholera vibrio, responsible for the disease.

Although the medical profession in general did not believe in the contagion of cholera, the Quebec population feared it like the plague and in the face of its rapid spread felt that close contact was a danger factor in its propagation. The patients coming from Grosse Isle and destined for admission to the Marine Hospital crowded on to the *quai des Indes* and the *quai de la Reine* and had to pass right through the town before reaching the hospital, which was situated on the banks of the St. Charles river, at the end of St. Roch quarter. Because of the state of the wood-paved streets and the steep slopes to be climbed, this journey constituted a "stress" sufficient to prove fatal to the poor patients, especially the incipient cases and those disembarked in the algid state.

The Board of Health had therefore decided to instal a temporary hospital in the Customs House, situated right in the Champlain district. This building was at that time lodging the muni-

cipal police. The neighbouring population, terrified at the thought of having in their midst a constant source of infection capable of spreading the disease and likely to cost them dear, protested to Dr. J. Douglas, who paid no attention to their protests. On the evening of July 11, at about 9 p.m., 2,000 inhabitants of the Champlain district carried by assault the police barracks which were about to be transformed into a cholera hospital, and demolished doors, windows, staircases, in fact, everything but the walls. The few policemen stationed there took to their heels. Mr. Symes, the sanitary inspector, rushed up and tried to restore order, but was jostled and beaten and had to retire with a bleeding face. The Board of Health had the building repaired, regretted the disturbance, and decided that the Marine Hospital, situated somewhat outside the town, should house the cholera cases, and that the town should pay for any citizens admitted.

TREATMENT

The methods of treating cholera at this time were very varied, especially as regards drugs. Dr. J. Painchaud, the doyen of Quebec medicine, gave advice to the population in the *Canadien* of July 13 and the *Journal de Québec* of July 12. It was considered at this time that certain symptoms now recognized as part of the disease were only premonitory. If they were treated at once, the disease could be prevented. This is what he said:

> "The precursory symptoms of cholera are a sudden malaise, a tendency to vomit, colic and irritant stools; if the latter tend to be whitish and like soapflakes, the danger is great and there is no time to be lost. Take what your doctor has already given you, and do exactly what he has advised, until he can get to you. If the symptoms get less, and particularly if the stools become darker, you are safe and you can leave your doctor free to rush to others in greater danger."

The Central Board of Health, Montreal, had laid down the general lines of treatment of cholera, and especially of the "precursory" signs (borborygmi, flatulence, a sensation of heat or repletion in the stomach, colic, a bad taste in the mouth, nausea), leaving the general practitioner to manage the case as he thought best once the diagnosis was confirmed. The following prescription was recommended from the start:

Tr. Zingib.	aa ℥ i
Tr. Piperis rubri	
Syr. Zingib.	℥ i

One teaspoon every half hour.

If diarrhœa was present, 15 drops of laudanum or one teaspoonful of elixir of paregoric in a little water was added, or alternatively the patient was to eat "a piece of opium præparata the size of a marble." If the onset was sudden, a mustard plaster was to be applied between the shoulders and in the epigastrium, while the feet were kept for 20 to 30 minutes in hot water containing a teaspoonful of mustard. The patient was to be put to bed, and covered up well with hot water bottles and hot flannels around his feet and body; in other words, copious sweating was to be induced. Purgatives, mineral water, alcoholic beverages, and especially brandy were to be avoided (*Journal de Québec*, July 21).

One young Quebec doctor, Dr. Rémi Cayer, used a preparation with a basis of aromatic tincture of iron chloride, which gave much publicized results. A merchant he had met in the United States and who had lived in India had extolled the virtues of this preparation to him. This young doctor at the bidding of his friends finally disclosed his preparation, and published the formula and the results in a childishly pompous article in a Quebec paper. Dr. Painchaud, then a visiting physician at the Marine Hospital, first commended him and promised to try out the preparation on cases of cholera in the hospital, following Dr. Cayer's instructions closely. After a few days, he hastened to publish the results of his trials in the same paper. Of the seven patients admitted on the 18th, five were dead; of the seven admitted on the 19th, all were dead. The patients had not responded any better to this preparation than to any other, and Dr. Painchaud seized the occasion to rail against what he called the charlatan's methods of his young colleague who "believed that he had found the sovereign remedy for cholera."

However, the *Journal* on July 25 printed as its headline "Has the cure for the terrible scourge of cholera at last been found?" and described the whole story of Dr. Cayer's famous remedy and the bitter remarks of Dr. Painchaud. Meanwhile, Cayer's remedy had travelled around, and laudatory letters arrived from Montreal, Charlesbourg, Beauharnois and elsewhere. In Montreal, Madame Lalonde, the famous singer, was attacked by extremely severe cholera and seemed likely to die. Dr. Picault, assisted by Dr. McCulloch, gave her Cayer's remedy and in a few hours she was better and in a few days convalescent (*Montreal Herald*, July 30).

Dr. von Iffland shared Dr. Cayer's view on the value of ferric chloride, but he pointed out to the public that this was not a scientific discovery but the use of a well-known method, utilized the preceding year in India and Teheran by Dr. Charles Bell, who had even reported the good results in England at the Manchester Royal Infirmary. Dr. Cayer confined Dr. von Iffland's statement, but added that he had personally improved the efficacity of the drug by adding a carminative, oil of anise. Dr. Cayer considered that the addition of oil of anise to his "mixture", as he called his prescription, converted the latter into "a powerful tonic, a severe astringent to the intestines, and a potent agent on the blood, and kept the patient's circulation free. The carminative has the effect of softening the violent action of tincture of ferric chloride on the stomach, and

preventing this organ from rejecting it by vomiting." (*Journal de Québec,* July 19).

These letters and invectives published in the press by the doctors helped to create some uneasiness in the population, and to arouse more fear of this disease which sometimes killed with lightning rapidity. In the United States, the disease was almost universal and the reports from the big cities such as New York, Philadelphia, St. Louis and Cincinnati were no more encouraging.

Since the appearance of the disease in Quebec, whole families had fled from the polluted atmosphere to the country. The doctors had however remained, and the Board of Health was pleased to recognize their devotion at the end of the epidemic, in spite of their dissensions on therapy. The undertakers had also remained at their posts. It is reported that in the Champlain district, which was the hardest hit, one of these gentlemen kept one of his hearses permanently available, ready harnessed and parked in the street. His horses, with their heads in a nosebag, were ready for the coachman, who answered any summons. The complaints about this macabre practice led to the withdrawal of the carriage and horses into the stables.

The cholera disappeared in mid-September. It had cost the City of Quebec £6,000. The work of the Board of Health and the hygiene measures taken by it had played a large part in checking its spread and its grave consequences. Nevertheless, 1,185 persons were buried in the cholera cemetery of Chemin St. Louis and the Catholic and Protestant Cemeteries of the Vacherie near to the Marine Hospital.

The disease reappeared on three further occasions in Quebec in 1851, 1852 and 1854, but its visits were brief. The epidemic of 1851 went by almost unknown to the population, and it was only at its end, when 280 persons had died, that its visit to Quebec was made public.

LE CHOLERA A QUEBEC EN 1849

SYLVIO LEBLOND, M.D., *Chicoutimi, Que.*

LE CHOLÉRA fit plusieurs apparitions au Canada. Quelques-unes des épidémies entrèrent au pays par Québec, qui était alors le port d'arrivée de tout navire qui venait d'Europe. Ce fut en 1832, que le choléra fit son entrée à Québec venu de Dublin sur le "Carrick." Il apparut sur la rue Champlain comme toutes les autres épidémies. Il fit 3,451 victimes dans la ville dont la population ne dépassait pas 30,000 âmes.

En 1834, nouvelle épidémie de choléra. Celle de 1832, avait déterminé le Gouvernement à construire à Québec, un Hôpital de Marine et des Emigrés dans le but de traiter et secourir les marins et les immigrants qui n'avaient d'autres endroits pour se loger que les maisons de pension du Quartier Champlain où ils étaient mal logés et surtout mal soignés, s'ils étaient malades.

L'hôpital encore incomplet ouvre quand même ses portes en 1834, pour recevoir les cholériques. Une fois l'épidémie disparue, il ferme en octobre 1834; les travaux se continuent et en mai 1835, il est officiellement inauguré. Cette année-là, 1834, le choléra fait 2,509 victimes à Québec. Il reparait de nouveau en 1849, et il fait mourir 1,185 Québecquois.

Cette épidémie de 1849 devient intéressante à cause de l'évolution des idées médicales de l'époque et de l'énoncé, dans certains milieux, de la contagiosité de la maladie, fait que l'on avait nié jusque-là. On croyait que la variole, le typhus, la typhoide étaient des maladies contagieuses qui se transmettaient par contact d'individus ou d'objets, mais on niait cette propriété au choléra qui n'apparaissait qu'à cause de changements inconnus, dans l'atmosphère. Quelques-uns même l'attribuaient à une surcharge de celle-ci en ozone et en profitaient pour expliquer chimiquement les bons effets du soufre et du charbon de bois contre le choléra.

Cette année-là le choléra nous vient des Etats-Unis par Kingston. Il n'apparut que très tard relativement à la Grosse-Isle (Quarantaine) alors même qu'il était à son déclin à Québec même. Québec était le port d'entrée de tous les navires venant d'Europe, et tout navire devait se soumettre à l'inspection à la Quarantaine de la Grosse-Isle, en saison de navigation. L'Inspecteur avait ordre de se servir d'armes à feu, si nécessaires pour faire obéir un capitaine récalcitrant.

Le courant d'émigration venant des Iles Britanniques et surtout d'Irlande où sévissait la famine, était très fort. En 1848 l'Europe avait été envahi par le choléra et en 1849 il régnait encore en Angleterre et en Irlande, en particulier. On espérait bien à Québec qu'il ne traverserait pas les mers.

Le Dr. Painchaud, en décembre 1848, avait donné une conférence publique sur le "Choléra Asiatique" qu'il terminait ainsi:

> "Si le choléra est en Angleterre, il aura bien le temps de s'éteindre avant que les bâtiments marchands partent pour le Canada. Quand même le choléra serait à New York, il ne peut passer nos lignes durant l'hiver; il pourra bien parcourir toute l'Amérique Méridionale, mais il sera éteint avant la fonte des neiges. Cependant la chose est possible, et c'est à nous tenir sur nos gardes." (*Le Journal de Québec*—4 janvier 1849).

Les conditions hygiéniques étaient bien pauvres à Québec pour empêcher ou prévenir la propagation de la maladie. On n'avait ni aqueduc ni égoûts. On puisait l'eau dans des sources qui étaient nombreuses, dans des puits ou encore dans le fleuve St-Laurent, soit à l'embouchure de la rivière St-Charles ou sur les quais. Toute une organisation de charroyeurs d'eau s'était faite qui distribuait l'eau à une population d'environ 37,000 habitants. Les égoûts et les déchets s'ac-

cumulaient dans les cours, les écuries ou dans les rues, dégageant, en certains quartiers, des odeurs parfois irrespirables. Quelques propriétaires s'étaient installés des tuyauteries à eux qui apportaient au fleuve les déjections mais elles aboutissaient aux endroits mêmes où on puisait l'eau potable pour la population.

C'est à la suite du choléra de 1849, que le Bureau de Santé de Québec, forma une proposition et insista sur l'urgence d'établir à Québec, sol rocailleux se prêtant mal aux absorptions, un aqueduc qui apporterait l'eau à la maison, et des égoûts. On alla chercher l'eau à Lorretteville, à la réserve indienne, et en 1854 l'aqueduc entrait en opération. C'était déjà une grosse amélioration. Il faut attendre un peu plus tard pour avoir les égoûts, cependant.

Le choléra apparut à Québec le 4 juillet, à la Basse-Ville au Quartier Champlain. Ce Quartier était situé au pied du Cap entre celui-ci et le fleuve St-Laurent. Il ne contenait qu'une seule et longue rue, étroite, construite des deux côtés. Une population pauvre et nombreuse y vivait à l'année, augmentée pendant la saison de la navigation par des marins et des émigrés qui s'y entassaient.

Au bout de cette rue, à un endroit qu'on appelait le cul-de-sac de la Basse-Ville on cueillait l'eau potable. L'état de malpropreté qui régnait là était suffisant pour les partisans de la non-contagiosité, pour vicier l'atmosphère et favoriser le développement du choléra. D'autres, les contagionnistes, attribuaient l'éclosion de la maladie à la présence de paillassons que des voiliers ayant souffert de la maladie, avaient jeté par-dessus bord et qui étaient venus se déposer près du cul-de-sac de la Basse-Ville, là où on puisait d'eau à boire. La maladie se propagea très vite dans ce Quartier et dans toute la ville. Un nommé McGill, un cordonnier en fut la première victime. Il mourut en quelques heures de maladie, le 4 juillet.

Un Bureau de Santé Central avait été formé à Montréal, par acte gouvernemental (Act-12 Vict. ch.8). Il était composé de MM. les Drs. Wilfred Nelson, Guillaume Deschambault et Robert Ley MacDonnell, de MM. Olivier Berthelot, William Workman, John James Day et Moses J. Hayes. Le Dr. Aaron H. David en était le Secrétaire. Il avait édicté des ordonnances et des règlements, insistant surtout sur la propreté, le nettoyage des rues, des cours et des dépendances. Chaque bureau local devait lui faire rapport.

A Québec, un Bureau de Santé existait, depuis 1832, qui reprenait fonction, chaque année, suivant les circonstances. Il avait été très actif en 1832, 1834 et 1847. Il avait acquis une expérience considérable, étant donné qu'on avait conservé autant que possible les mêmes hommes. Furent nommés cette année–là MM. Légaré, Sewell, Dr. Morrin, Dr. Nault, M. Boxer, Dr. Paradis, Dr. Joseph Painchaud et le Dr. James Douglas. La plupart avaient siégé sur ce Comité pendant l'épidémie de typhus de 1847. M. Robert Symes fut nommé inspecteur sanitaire au salaire de 10 sh. par jour et F. X. Garneau, qui préparait alors l'Histoire du Canada, qui le rendra célèbre, en était le secrétaire. Le Dr. Morrin en était le président.

Ce Dr. Jos. Morrin était un homme très occupé, doué d'un très grand sens social. Il venait de fonder à Québec une Ecole de Médecine. Il s'occupait activement de l'Asyle de Beauport avec le Dr. J. Douglas et il était échevin de la Cité de Québec. L'Université Laval transforma son Ecole de Médecine en Faculté de Médecine et il lui laissa à sa mort une gratification suffisante qu'on appelle encore le Prix Morrin. Ce Bureau de Santé fut très actif. Il organisa les visites domiciliaires et quatre médecins étaient désignés pour ces visites avec devoir de rapporter tout cas de maladie, choléra ou autre. Ce furent les Drs. Bardy, Wolf, Robitaille et Carrier. Ces rapports empreints d'un sens social très avancé, ont contribué fortement à assainir la ville, les rues, les quais de la vieille cité, à promouvoir l'installation de l'aqueduc, des égoûts, et même l'amélioration de l'état financier de la ville.

CONTAGIOSITÉ OU NON CONTAGIOSITÉ

L'opinion générale, à cette époque, était que le choléra n'était pas une maladie contagieuse au même titre que la variole, la typhoide ou le typhus. Le contact ou le voisinage avec les cholériques n'apportait pas le choléra et on donnait comme exemple les médecins, les infirmières, les prêtres, les parents qui côtoyaient les malades sans prendre la maladie, contrairement au typhus, qui deux ans auparavant, avait fait de nombreuses victimes chez les médecins, les prêtres, et le personnel hospitalier, notamment à la Grosse-Isle.

Cependant, de temps à autre, une voix s'élevait qui criait: "Attention." En juillet 1849, un alarmiste qui signait "Delta" avait écrit dans la *Gazette* et le *Mercury* où il disait:

> "Je suis alarmiste et je voudrais alarmer tout le monde, le Bureau de Santé ne voulant pas le faire, assez pour qu'on ne s'exposât pas sans nécessité au danger. La peur ne donnera pas le choléra, mais la peur y prédisposera, et si avec cela vous allez au-devant de la maladie, vous pourrez la contracter et la contracterez probablement."

Ce pauvre "Delta" se vit vilipender par des médecins, des citoyens, les journaux et un bon jour il décida de ne plus continuer la polémique; et un article humoristique des funérailles journalistiques de ce Monsieur, fut publié dans le *Mercury de Québec*. (28 août 1849). On l'accusait d'avoir engendré la peur chez un certain nombre de citoyens qui prirent ainsi le choléra et en moururent.

Contre la contagiosité on apportait aussi l'argument que des individus en parfaite santé n'ayant eu aucun contact avec des cholériques, étaient pris brusquement, de crampes, de diar-

rhée profuse, d'algidité et liquidé en quelques heures, tandis que d'autres, des convoyeurs de morts, résistaient à toute infection, que des hôpitaux situés en pleins quartiers populeux à Londres et ailleurs et hébergeant des cholériques n'avaient pas transmis la maladie et qu'on avait vu peu de cas de choléra parmi le personnel médical et autre.

Le Dr. John Hall, chirurgien de Québec, publie un long article dans le *Mercury de Québec* du 18 août, contre la contagiosité du choléra et il termine en rapportant les conclusions du Bureau de Sante de la ville de New York, parues dans le *New York American Journal* de juillet de la même année.

"The undersigned believe that the cause of the disease exists in the atmosphere and that the whole of the community are at present more or less under the influence of the peculiar condition of the atmosphere . . . and in this way predisposed to the disease. To develop the disease, however, existing causes are necessary, and these are to be found in all those things which have a tendency to disorder the bowels. With regard to the peculiar condition of the atmosphere which predisposes to the disease, we know nothing. Human skill and agency, therefore, can do nothing in meeting this difficulty. The existing causes, on the contrary are in a great measure under our control, and by properly guarding against these, much, very much may be done in obviating the development and extension of the disease", et c'est signé: John Beck, M.D., Sam'l W. Moore, M. David, Joseph Smith.

On croyait donc généralement que la maladie était dans l'air, l'atmosphère, et qu'elle ne se développait qu'à l'occasion de certaines causes favorisantes, existant chez l'individu, dans son comportement ou dans son entourage atmosphérique. La malpropreté, les odeurs méphitiques et nauséabondes, la fatigue, l'inquiétude, la peur, la privation d'aliments et surtout l'usage de boissons alcooliques provoquaient à coup sûr l'apparition de la maladie. Il y avait des sceptiques cependant, qui croyaient que toute cette campagne favorisant le nettoyage des rues, des cours n'étaient propres qu'à effrayer les gens.

Le *Quebec Morning Chronicle* du 23 juillet disait:

"With all respect for our contemporaries . . . we state plainly, dogmatically and confidently, that neither diet nor cleanliness have anything to do with the matter; whether chloride of lime, of zinc, burning barrels of tar or draining cesspools are vain and useless in the open air. The drains and sewers smell no worse this year than last, vegetable matter decays not more speedily this summer than it did in July 1848, the poor are not dirtier, nor the rich cleaner than they ever have been; poverty and sloth produce dirt now as ever, and wealth revels on clean linen and drops asleep on comfortable beds and well aired bed rooms at present as they have been wont; yet both classes are seized with cholera, and the disease shows little favour. . . ."

Le Dr. Von Iffland, médecin distingué de Québec, attaché à cette époque à l'Asyle de Beauport, qu'avait fondé, en 1845, le Dr. James Douglas, adresse au Canadien de Québec, une longue lettre sur la "non-contagiosité" du choléra, que ce journal publie le 18 juin, quelques semaines avant l'apparition de la maladie. Pour lui le choléra n'est pas contagieux car s'il l'était la race humaine n'existerait plus. Cette maladie, à l'encontre de la variole, peut attaquer plusieurs fois le même individu et si elle était contagieuse elle ne disparaitrait que faute de sujet.

Toutes les maladies épidémiques, explique-t-il, tirent leur origine d'une même cause, à savoir, l'état de l'air. C'est la meilleure solution qu'on puisse en donner bien que l'on ne sache pas quelles sont les conditions de l'atmosphère qui favorisent ou produisent ces maladies. Il est un fait c'est que ces épidémies apparaissent surtout lorsque l'atmosphère est humide et chaude, et il ajoute: "Les changements de l'air, à ces époques, doivent nous rester à jamais inconnus."

Il ne savait pas encore, le Dr. Von Iffland, pas plus lui que bien d'autres, que déjà, à Paris, Pasteur était en train de trouver une réponse à ces inconnus, réponse qui détruiraient toutes ces belles théories, et qu'en 1885 Koch montrerait au monde le vibrion cholérique, le responsable de la maladie.

Si la profession médicale en général ne croyait pas le choléra contagieux, la population de Québec, le craignait comme une peste, et constatant son expansion rapide, pensait bien qu'un contact rapproché était dangereux pour sa propagation. Les malades venant de la Grosse-Isle et devant être hospitalisé à l'Hôpital de la Marine, abondaient au quai des Indes ou au quai de la Reine et devaient traverser toute la ville pour arriver à cet hôpital qui était situé sur les bords de la rivière St-Charles, aux confins du Quartier St. Roch. Etant donné l'état des rues, les côtes assez abruptes à gravir, pavées en bois, ce voyage constituait pour ces pauvres malades, et en particulier pour les cholériques à venir, surtout ceux qui seraient débarquées à la période d'algidité, un "stress" suffisant, pour les faire mourir.

Le Bureau de Santé, avait donc décidé d'installer un hôpital temporaire dans l'edifice de la douane situé en plein Quartier Champlain. Cet édifice logeait alors la police municipale. La population du Quartier, effrayée d'avoir en plein milieu d'eux, un foyer d'infection constant qui pourrait propager la maladie, à qui elle avait déjà payé un fort tribut, proteste auprès du Dr. J. Douglas qui reçoit mal ses remarques.

Le 11 juillet au soir, vers 9 hr, 2,000 habitants de ce quartier Champlain, prennent d'assaut la caserne de la police qu'on veut transformer en hôpital de cholériques, démolissent portes, fenêtres, escaliers, tout, excepté les murs. Les quelques agents de police qui y étaient stationnés prennent la fuite. M. Symes, l'inspecteur du Bureau de Santé, qui accourt, essaye de rétablir l'ordre. Il est bousculé, battu et il doit se retirer la face en sang. Le Bureau de Santé fait réparer l'édifice, déplore les incidents survenus et décide que l'Hôpital de la Marine situé, un peu en dehors des limites de la ville, hébergera les cholériques, et que la ville paiera les frais des citoyens hospitalisés.

TRAITEMENT

Les modes de traitement du choléra à cette époque étaient très variables, surtout en ce qui concernaient les médicaments. Le Dr. J. Painchaud, doyen des médecins de Québec, donnait dans le *Canadien* du 13 juillet et le *Journal de Québec* du 12 juillet, les conseils d'usage à la population. On admettait alors que certains symptômes considérés aujourd'hui comme faisant partie de la maladie, n'étaient alors que les signes avant-coureurs de l'infection. Traités dès lors on pouvait éviter l'atteinte de la maladie. Voici ce qu'il disait:

"Les symptômes avant-coureurs du choléra sont un malaise subit, une tendance à vomir, des coliques et des selles épineuses; si ces dernières tirent sur un blanc approchant celui de la savonnure, le danger est grand, et il n'y a pas de temps à perdre. Prenez alors ce que votre médecin vous aura donné d'avance; faites exactement ce qu'il vous aura recommandé, jusqu'à ce qu'il soit auprès de vous. Si les symptômes diminuent, et si les selles, surtout, sont d'une couleur plus foncée, vous êtes en sûreté, et vous pouvez laisser votre médecin courir à d'autres plus en danger que vous."

Le Bureau Central de Santé de Montréal avait édicté les données générales du traitement du choléra, surtout des signs "avant-coureurs" (grondement de boyaux, flatuosité, chaleur ou réplétion dans l'estomac, coliques, mauvaise bouche, nausées), laissant au médecin traitant la conduite thérapeutique qu'il jugera bon d'utiliser une fois la maladie confirmée.

Voici ce qu'on recommandait dès le début:

Teinture de gingembre Teinture de poivre rouge	i ℥ $\overline{aa}$
Sirop de gingembre	i ℥

1 c. à thé toutes les demi-heures

Si la diarrhée existe on ajoute 15 gouttes de laudanum ou une c.à thé d'élixir parégorique dans un peu d'eau, ou encore "manger un morceau de préparation d'opium de la grosseur d'un marbre." Dans les cas d'apparition brusque et subite, on prescrivait des applications d'emplâtre de moutarde entre les deux épaules sur le creux de l'estomac, les pieds dans l'eau chaude contenant 1 c.à thé de moutarde pendant 20 à 30 minutes. Mettre le malade au lit, le couvrir abondamment, mettre des bouteilles d'eau chaude et des flanelles chaudes autour des pieds et du corps; en somme provoquer une "suerie" abondante. Eviter les purgatifs, les eaux minérales, les boissons alcooliques en particulier le brandy. (*Journal de Québec*, 21 juillet).

Un jeune médecin de Québec, le Dr. Rémi Cayer utilisait une préparation à base de tr de muriate de fer aromatisée, qui lui donnait des résultats dont on parlait beaucoup. Un négociant, rencontré aux Etats-Unis, et qui avait vécu aux Indes, lui avait vanté les bons effets de ce médicament. Ce jeune médecin, sur les instances de ses amis, finit par produire son médicament, et dans un article d'une pompe enfantine publie dans un journal de Québec, sa formule et ses résultats. Le Dr. Painchaud, alors médecin visiteur à l'Hôpital de la Marine, le louange fort d'abord et lui promet d'essayer son médicament en suivant bien les indications données par le Dr. Cayer, chez des cholériques de l'Hôpital. Il ne tarde pas au bout de quelques jours de publier dans le même journal les résultats de son essai. Des 7 cas admis le 18-5 sont morts. Des 7 cas admis le 19-7 sont morts. Ses malades n'ont pas répondu mieux qu'à toute autre médication et il en profite pour déblatérer contre ce qu'il appelait les façons charlatanesques de son jeune confrère qui "croyait avoir trouvé le grand remède contre le choléra."

Le *Journal* du 25 juillet d'ailleurs écrivait en tête de son reportage: "Le choléra: Le remède à ce fléau redoutable est-il enfin trouvé?" . . . et il racontait toute l'histoire du fameux remède du Dr. Cayer et des remarques acerbes du Dr. Painchaud."

Pendant ce temps le remède Cayer avait fait son chemin et des lettres louangeuses arrivaient de Montréal, de Charlesbourg, de Beauharnois, etc. A Montréal Mme Lalonde, la célèbre cantatrice est atteinte d'un choléra excessivement grave. On croit qu'elle va mourir. Le Dr. Picault assisté du Dr. M'Culloch, lui administre le remède Cayer. En quelques heures elle se remettait et au bout de quelques jours elle était convalescente. (*Montreal Herald* 30 juillet).

Le Dr. Von Iffland partage l'opinion du Dr. Cayer sur la valeur du fer muriatisé, mais il apporte au public le fait que l'on n'est pas en présence d'une découverte scientifique, mais d'une méthode déjà connue, utilisée aux Indes et Téhéran l'année précédente par le Dr. Charles Bell, qui d'ailleurs en avait annoncé les bons effets en Angleterre au Royal Infirmary de Manchester. Le Dr. Cayer confirme d'ailleurs cette assertion du Dr. Von Iffland, mais il ajoute que ce qui augmente l'efficacité de ce médicament c'est que, lui, il y ajoute un carminatif l'huile d'anis. Le Dr. Cayer considérait que l'addition d'huile d'anis à sa "mixtion," comme il appelait sa prescription, faisait de celle-ci "un tonique puissant, un astringent sévère sur les intestins, un agent fort sur le sang, et conservait au patient sa circulation libre." "Le carminatif avait pour effet d'adoucir l'action violente que la teinture de muriate de fer opère sur l'estomac et empêcher que cet organe ne la rejette par le vomissement.' (*Journal de Québec*, 19 juillet).

Ces lettres et ces invectives entre médecins par la voie des journaux contribuaient à créer un certain malaise dans la population et entretenir la peur contre cette maladie qui parfois tuait avec une rapidité foudroyante. Aux Etats-Unis la maladie sévissait un peu partout et les rapports qui arrivaient des grandes villes comme New York, Philadelphie, St-Louis, Cincinnati, n'étaient pas plus encourageants. Dès l'apparition de la maladie à Québec, des familles entières

déménagèrent à la campagne, fuyant une atmosphère polluée. Les médecins cependant, restèrent sur place et le Bureau de Santé se plut à reconnaître leur dévoûment à la fin de l'épidémie, malgré leurs dissensions thérapeutiques.

Les croquemorts aussi restèrent au service de la population. On rapporte que dans le Quartier Champlain, le plus affecté, un de ces messieurs gardait en permanence un de ses corbillards (hearses) à la disposition, toujours attelé, et stationné en pleine rue. Ses chevaux, la tête dans leur mangeoire, attendaient le cocher qui répondait aux appels. Des plaintes contre cette macabre coutume firent entrer dans ses écuries, la voiture et son attelage.

Le choléra disparut vers la mi-septembre. Il avait coûté £6,000 à la Cité de Québec. Le travail du Bureau de Santé, les mesures hygiéniques prises contribuèrent fortement à en atténuer l'expansion et les effets néfastes. Quand même 1,185 individus y laissèrent leurs os, dans le cimetière des cholériques du Chemin St-Louis, et les cimetières catholiques et protestants de la Vacherie près de l'Hôpital de la Marine.

La maladie fit encore trois apparitions à Québec en 1851, 1852 et en 1854, mais elles furent de courte durée. Celle de 1851, passa à peu près inconnue de la population et ce n'est qu'à la fin de l'épidémie, qui fit mourir environ 280 individus, qu'on fit connaître son passage à Québec.

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MEDICAL SOCIETIES

CANADIAN PSYCHOANALYTIC SOCIETY

Election.—Dr. Hans Aufreiter, training analyst of the Vienna Psycho-Analytical Society and Institute, was elected a member of the Canadian Psychoanalytic Society on May 28, 1954.

Scientific meeting (Montreal, June 13, 1954).—A paper was delivered on "The Problem of Transference," by Dr. Daniel Lagache, president of the Société française de psychanalyse and professor of Psychology at the Sorbonne. A few of the author's original contributions deserve special mention. A distinction was drawn between the analyst's transference on the analyzand and counter-transference, the latter being a response to transference; thus, both the analyst and the patient may exhibit at times attitudes due to counter-transference. Positive transference was defined as the type of doctor-patient relationship which promotes the treatment's progress; negative transference hinders the course of analysis.

Public meeting (Montreal, June 14, 1954).—The second public meeting of the Society was held at the Hôtel-Dieu Hospital and addressed by Dr. D. Lagache, who spoke on "The Results of Psychoanalytic Treatment." The guest speaker emphasized the following criteria in mental health: capacity of producing, tolerating and reducing tensions (needs), with satisfaction for the individual; capacity to realize one's possibilities; capacity to organize a plan of life; capacity to adjust one's aspirations; capacity to deal with other people; capacity to identify both with the conservative and the creative forces in society. Psychoanalytic cure is "a new health."

J. B. BOULANGER

CANADIAN DERMATOLOGICAL SOCIETY

The eighth annual meeting of the Canadian Dermatological Association was held at Harrison Hot Springs Hotel, B.C. on June 13 to 15 and at Shaughnessy Hospital, Vancouver, B.C. on June 16, 1954. Twenty-two members and 12 guests were present. The following papers were read: (1) Introductory Remarks—Dr. D. H. Williams, Vancouver. (2) A Case of Sporotrichosis—Drs. L. P. Ereaux, G. E. Craig, G. Kalz and F. Blank, Montreal—Read by Dr. C. J. Fournier, Vancouver. (3) Porphyria—Report of Two Cases—Dr. G. B. Sexton, London. (4) Vitamin D_2 in Dermatology—Dr. J. Grandbois, Quebec. (5) The Problem of Antibiotic Resistance of Staphylococci—Dr. G. Williamson, Ottawa. (6) Recent Addition to Failure in Therapy in Onychomycosis—Dr. R. Schacter, Toronto. (7) Preliminary Report on Laboratory Investigations in Vitiligo—Dr. K. G. Davidson, Winnipeg. (8) A Small Punch Biopsy Technique—Dr. D. H. Williams, Vancouver. (9) Cutaneous Leishmaniasis—Report of Three Cases—Dr. R. C. Smith, Toronto. (10) Impetigo Herpetiformis Recurrens—Dr. E. Gaumond, Quebec. (11) Kerato-Acanthoma and Related Epithelial Hyperplasias—Drs. P. Schopflocher and L. P. Ereaux, Montreal, Que. (12) Leprosy—Report of Three Cases—Dr. B. Kanee, Vancouver. (13) Contact Dermatitis from Platinum Metals—Report of a Case—Dr. C. S. Sheard, Stamford, Conn.

Following the annual dinner on Monday, June 14, Dr. G. S. Williamson of Ottawa presented a highly interesting, illustrated talk "Arctic Adventure," describing his trip to the Canadian Arctic in 1953.

On Wednesday, June 16 a clinical session was held at Shaughnessy Military Hospital, Vancouver, followed by luncheon at Shaughnessy Heights Golf Club.

The following officers were elected for 1954-1955: President: Dr. N. M. Wrong, Toronto, Ont.; Vice-president: Dr. J. P. Grandbois, Quebec, Que. Secretary: Dr. A. R. Birt, Winnipeg, Man.; Regional Secretary: Dr. R. C. Smith, Toronto, Ont.

A. R. BIRT

THE SEVENTH INTERNATIONAL CONFERENCE OF SOCIAL WORK

Toronto, June 27 to July 2, 1954

The Seventh International Conference of Social Work convened in Toronto on June 27, 1954, under the presidency of George F. Haynes. Eighteen hundred delegates from 48 countries registered for the Conference.

Approximately 350 delegates came from abroad, and it was noted that particularly strong delegations represented Asiatic and Western European countries. Canada's Dr. G. F. Davidson, Deputy Minister of National Welfare was President of the Canadian Conference of Social Work which met conjointly with the International Conference.

The promotion of social welfare activities through self-help and co-operative action was the theme which was carried throughout the five days of meetings, and the varied points of view from a wide representation of countries resulted in a realistic approach to problems under discussion. Various broad subjects concerning social welfare problems were introduced by special speakers at plenary sessions during morning or evening meetings. These subjects were further explored at subsequent panel discussions and finally, special aspects of these broad areas of interest were considered by a series of study groups which met in afternoon sessions.

As a fitting opening for the Conference, the Honourable Lester B. Pearson, Minister for External Affairs, Government of Canada, addressed the first plenary session on "The World We Live In." Mr. Pearson emphasized the importance of recognizing the rights and privileges of countries and individuals in these countries. He stated that citizens of all nations are proud of their heritage, and like to do things their own way; they generally recognize the problems which are paramount in their own locale and are anxious to help themselves. It is important that people be given a chance to help themselves before turning to others.

Other plenary sessions included the following subjects and speakers: "The Meaning of Self-Help in Social Welfare"—Alan Moncrieff, M.D., Nuffield Professor of Child Health, University of London, and Director of the Institute of Child Health. "Threats to Self-Help"—M. Georges Desmottes, Assistant Director, French Ministry of Health. "Co-operative Action and the World Community"—Norman Cousins, Editor, "The Saturday Review," New York City. "Self-Help in Modern Society"—J. F. deJongh, School of Social Work, Amsterdam, Netherlands. "Leadership for Self-Help"—A. N. Sinha, Minister for Finance, Government of Bihar, India.

A number of special meetings were also held by various associated agencies during the conference. The International Federation of Social Workers held a meeting to discuss the European Social Welfare Exchange Programme; whereas problems of particular concern to the International Conference on Non-Governmental Organizations Interested in Migration, were discussed by a special meeting of this group. Various church organizations also held meetings during the Conference.

The Conference was successful both regarding attendance and the intellectual contribution of the delegates. The varied experience from so many countries was both interesting and helpful to those dealing with similar problems in their own country or jurisdiction. Although some countries have more social or welfare problems of one kind or another than other countries, the feelings and reactions of individuals in all countries towards social assistance is essentially the same. People of every country, although in dire circumstances, still wish to keep their self respect and want to be treated as individuals. It was pointed out, however, that in some cases where individuals are diseased, otherwise disabled, or in extreme economic need, self-help is not possible. In such cases individuals must first have their health and confidence restored before they are able to get a new start.

The large number of physicians representing various health agencies who attended the Conference might be considered a healthy sign that the need for a closer working relationship between health and welfare problems is gaining recognition. This integration of health and welfare activities appears to be more pronounced in European and Asiatic countries. Possibly this has resulted because of the special problems of disease, hunger and want that exist in some of these areas, which have required the concerted efforts of health and welfare agencies to deal with them.

MISCELLANY

MOBILE EMERGENCY UNIT

The first of its kind in the world, Molson's Mobile Emergency Unit incorporates into a single self-contained vehicle all the latest power, communications, first aid, and kitchen equipment normally found in three or four separate relief or rescue vehicles.

An example of this Unit was recently presented to the public in Montreal by the president of Molson's Brewery Limited as a public service.

The Molson unit is divided into three sections, with electrical and electronic equipment in the front section, first aid facilities in the centre, and kitchen in the rear section.

The three sections are housed in a 42½-foot long trailer powered by a tilt cab tractor. The all-steel vehicle is electrically spot-welded and fully insulated. It was designed and built by Diamond T Motor Trucks Montreal Limited and is equipped with material purchased from Canadian suppliers.

The main equipment in the front, or electrical, section is: a two-way radio; a public address system for use as an aid in preventing panics, directing relief workers, or guiding people lost in the bush; two 1,000-watt searchlights; an Aldis signalling lamp; a 5,000-watt generator; a 1,000-watt portable generator, and auxiliary heater. A Bell Mobile Telephone system has been installed in the cab.

The centre, or hospital section, 25 feet long, contains: complete emergency first aid supplies, an operating table, a complete set of standard surgical instruments, 10 folding-type aluminum stretchers, 3 dozen blankets, complete resuscitation equipment, 2 folding beds, 2 folding tables, a supply of drugs and intravenous anæsthetics, and all other necessary medical equipment.

The stainless steel kitchen, or rear, section is equipped with a refrigerator, complete cooking facilities and a 200-gallon water tank. Up to 200 cups of coffee per hour can be dispensed from a serving window. In addition, hot and cold full-course meals or snacks can be prepared.

Molson's Mobile Emergency Unit also carries all other necessary rescue work equipment such as fire extinguishers, axes, shovels, crowbars, rope, wire, flashlights, flares and portable radio sets.

SPECIAL CORRESPONDENCE

The London Letter

(From our own correspondent)

OBSTETRICAL SILVER JUBILEE

The Royal College of Obstetricians and Gynæcologists has been celebrating the silver jubilee of its foundation in 1929. The traditional reception, conferment of honorary Fellowships and banquet have been held to commemorate the 25th anniversary of an institution which met with the utmost opposition during its period of gestation. At the time of its registration the College consisted of only the nine signatories to the articles of Association. Today it has almost 400 Fellows and over 1,000 members, many of whom come from the British Commonwealth overseas. The intriguing story of these formative years is told in a commemorative history of the College, *Twenty-five Years*, written by Sir William Fletcher Shaw who for five years was honorary secretary of the committee which brought the College into being, and then honorary secretary of the College until 1938 when he was elected President. The celebrations are also

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being made the occasion for a Silver Jubilee Building Appeal for £400,000 to allow the College to build new premises in Regent's Park. It has outgrown its present home in Queen Anne Street (the gift of Professor Blair Bell, the first president of the College), and it is felt that unless more commodious premises are obtained future development of the College will be severely curtailed.

GERONTOLOGISTS IN CONCLAVE

The gerontologists have come and gone, having, literally, put old age and its problems in the headlines of the national press for a week, and having demonstrated to the medical profession the many gaps in our knowledge of the fundamental basis of the aging process. The occasion was the third congress of the International Association of Gerontology which met here on July 19 to 23 under the presidency of Dr. J. H. Sheldon of Wolverhampton. Some 600 delegates, 350 of whom came from 42 countries overseas, attended and had a selection of 244 papers from which to pick and choose. The programme covered the whole gamut of old age in health and disease—from the biology of the aging cell to the etiology of osteoarthritis, and from ideally nutritive bread in New York to the employment problems of the aged. In order to render the rarefied atmosphere induced by the biologists more adaptable to the hard facts of life, delegates were given ample opportunity of seeing for themselves the various schemes for ameliorating the lot of the elderly which have been developed over here during the postwar years.

A NOTABLE CENTENARY

This month the Royal Hospital and Home for Incurables is celebrating its centenary. In a commemorative article in *The Practitioner*, Dr. Verna Kendall, medical officer to the establishment, outlines the history of the institution. It was founded in 1854 by the Rev. Andrew Reed, D.D., who was also responsible for founding four other great charities, including three orphanages and an institution for mental defectives. The Home was originally established in a rented house, with four inmates. The present premises in Putney, a suburb on the southwest side of London, were acquired in 1865, and there are now 260 residents. In addition, small annuities are paid to 250 pensioners living in their own homes. It is still a voluntary hospital as it was disclaimed by the Minister of Health in 1948, on the grounds that it is not a hospital within the meaning of the National Health Service Act, because it has no operating theatre. In other respects it is a self-contained unit, providing full nursing care, general-practitioner and consultant services, dentistry, physiotherapy, radiography, occupational therapy and dispensing. The criterion for admission is total incapacity to earn a living. Patients known to be suffering from carcinoma, pulmonary tuberculosis, epilepsy and unsoundness of mind are not admitted, but responsibility is accepted for those who develop any of these conditions after admission.

HOSPITALS IN COURT

In the House of Commons the Minister of Health was recently asked how much money his department has paid each year, since the health services were nationalized, in damages for neglect or inefficient medical or nursing treatment. In reply, he stated that the total compensation payments of all kinds made by National Health Service hospital authorities in England and Wales since the introduction of the Service were as follows:

July 5, 1948 to March 31, 1949	£ 7,560
Year ended March 31, 1950	£ 23,636
Year ended March 31, 1951	£ 38,556
Year ended March 31, 1952	£106,574
Year ended March 31, 1953	£152,590

These payments result very largely from claims for damages for neglect and inefficient medical or nursing treatment.

London, August 1954. WILLIAM A. R. THOMSON

OBITUARIES

DR. HENRY A. BEATTY died at Toronto, Ont., on June 29 at the age of 79. Born in Thorold, Ont., he graduated from the University of Toronto School of Medicine in 1897. In the same year he went to London, Eng., for postgraduate study then on to Vienna for further studies in eye, ear, nose and throat diseases. Returning to London he joined the staff of the Poplar Accident Hospital and in 1901 became senior house surgeon at Westminster. The following year he returned to Toronto and joined the Canadian Pacific Railway as chief surgeon of the Ontario Division. By 1910 Dr. Beatty had become chief surgeon and medical officer, Eastern Lines, and six years later was appointed to the same position for all lines. During this period he also became a member of the surgical staff of the Toronto Western Hospital, and in 1924 he was made surgeon-in-chief as well as assistant professor of clinical surgery at the university. In 1933 Dr. Beatty became consulting surgeon and a life governor of the hospital. In 1946, after 44 years' service, he relinquished his posts but remained as a consultant. Dr. Beatty was a member of the Royal College of Surgeons and a fellow of the American College of Surgeons.

DR. JOHN D. CHRISTIE, 80, died at Toronto, Ont., on July 8. Born in Manchester he graduated from the University of Toronto in 1905. After practising in Saskatchewan and at Mattawa, Ont., he established his practice in Toronto where he remained for 23 years. Since his retirement 13 years ago Dr. Christie had resided in Leaside. He is survived by his widow and three daughters.

DR. FREDERICK F. A. CORBETT died at his home in Regina, Sask., on July 3. Born at Five Islands, Colchester County, N.S., in 1870, he graduated from Mount Allison University and obtained his medical degree at McGill University in 1896. After practising first at Parrsboro, N.S., he took postgraduate study in Berlin, London and Edinburgh where be became a fellow of the Royal College of Surgeons in 1911. That same year Dr. Corbett moved to Regina where he practised until his retirement in 1946. He pioneered in cancer research when the first clinics were established at the General Hospital in Regina. He was also consulting surgeon for the cancer clinic in the Grey Nuns Hospital until his retirement. He was a member of the American College of Physicians and Surgeons and the College of Surgeons in Canada. Dr. Corbett is survived by his widow, two daughters and one son.

DR. ROY J. COYLE died in Windsor, Ont., recently at the age of 55. Born on Wolfe Island near Kingston, Ont., he received his medical degree from Queen's University. He took postgraduate studies at New York Nose and Throat Hospital and at St. Thomas Hospital, London, Eng., and thirty years ago established his practice in Windsor as an eye, ear, nose and throat specialist. On June 3 of this year he was honoured at Assumption College's first university convocation with an alumni award for his services to the College.

DR. GEORGE HOWARD FARQUHARSON, 73, of Calgary, Alta., died on June 12. A native of Mount Herbert, P.E.I., he graduated in medicine from the University of Western Ontario in 1908. Immediately upon graduation, Dr. Farquharson went west to Gleichen, Alta., where he practised for 45 years. He retired to Calgary last October. He is survived by his widow, one daughter and one son.

DR. DONALD THOMAS FRASER, 65, associate director of the Connaught Medical Research Laboratories, University of Toronto, died suddenly in Santiago, Chile, on July 20. An authority on preventive medicine and

immunology, he was on a tour of South American medical schools and schools of public health at the request of the World Health Organization. A native of Toronto, he graduated in medicine from the University of Toronto. Early in 1915 he went overseas serving first in Egypt with the 94th Field Ambulance unit. The following year in France he was awarded the Military Cross for extreme bravery at the Somme. He was wounded and sent back to Canada but returned to service with the British Expeditionary force in China where he was again commended for gallantry at Weihaiwei in 1918. On his return Dr. Fraser joined the newly created Connaught Laboratories as bacteriologist and became associate director in 1932. He was also head of the University's department of hygiene and preventive medicine and associate director of the school of hygiene. A Fellow of the Royal Society of Canada, he was a member of both the American and Canadian Public Health Associations, the Association of Immunologists and the Association of Epidemiologists. An authority in many fields, Dr. Fraser was especially well known for his knowledge of diphtheria immunization. He is survived by his widow, a daughter and two sons.

DR. MARSHALL E. GILLRIE, 91, died at Hamilton, Ont., in July. Born at Holland Landing, Ont., five years before Confederation, he graduated from Victoria College in 1888 and from the University of Toronto School of Medicine in 1889. He practised at St. Mary's for four years before establishing his practice in Hamilton 61 years ago. Dr. Gillrie was known for the many reminiscences of his life as a general practitioner when he made his rounds by bicycle half a century ago. He was president of the Hamilton Academy of Medicine in 1916. Dr. Gillrie retired 20 years ago. He is survived by his widow.

DR. J. LEON HOUDE of Rimouski, Que., died at the Hôtel-Dieu de Québec on June 13. Born in Lotbinière County, Que., in 1886 he graduated in medicine from Laval University in 1912. Dr. Houde practised in L'Islet, Que., for five years before serving with the Canadian Army in World War I. When the war ended he became interested in the field of public health and, after specialized studies in Toronto, he served as medical health officer of the lower St. Lawrence region until the time of his death. He is survived by his widow, one daughter and two sons.

DR. HILARION C. LEBLANC of Dieppe, N.B., died on May 14 at the Hôtel-Dieu Hospital, Moncton. Born at Pointe de l'Eglise, N.S., in 1864 he obtained his medical degree in Baltimore in 1891. After graduation he returned to his native province where he practised for several years. In 1920 Dr. LeBlanc moved to Moncton and established his practice as an eye, ear, nose and throat specialist. From 1922 until 1936 he was medical health officer for the schools of Northumberland, Westmorland and Kent Counties. On his retirement in 1947, Dr. LeBlanc moved to Dieppe. He is survived by his widow and ten children.

DR. FFORDE EDWARD MacLOGHLIN died on June 15 at the Mount Hamilton Hospital, Hamilton, Ont. Born in Hamilton 79 years ago, he graduated in medicine from the University of Toronto and returned to his native city to practise. For several years Dr. MacLoghlin served as president of the Hamilton Academy of Medicine. A son and a daughter predeceased him, his only son, Dr. Edward Gregg MacLoghlin, having been killed in Sicily in 1944 while serving as a captain in the RCAMC.

DR. ERNEST ALBERT MARTIN died recently in North Vancouver, B.C., at the age of 79. Graduating in medicine from McGill University in 1901, he practised at South Mountain and Kemptville, Ont. In 1909 he went west to Kelowna, B.C., and two years later moved to North Vancouver where he practised for more than 40 years. Dr. Martin had retired three years ago. His wife predeceased him.

DR. KENNETH MARDAYN MICKLEBOROUGH, 39, was instantly killed in a motor accident on July 17. A native of Ottawa, Ont., he graduated in medicine from the University of Toronto in 1941. The following year he joined the Royal Canadian Army Medical Corps and served overseas with the rank of captain. After the war Dr. Mickleborough established his practice in Ottawa and had been on the staff of Civic Hospital since that time. He was returning from a pre-dawn call at the Hospital when he was involved in a head-on collision. He is survived by his widow and one child.

DR. PERCIVAL PHILP, 54, died at Toronto, Ont., on July 21 after a lengthy illness. A native of Colborne, Ont., he served overseas during World War I with the Canadian Field Artillery. On his return to Canada he studied medicine at the University of Toronto, graduating in 1923. After postgraduate study in England, Dr. Philp practised in York Township and served on the staff of Christie Street Hospital. He was also a former chairman of the York Township Board of Education. He is survived by one daughter.

DR. ANTONIO POULIOT died at Quebec, Que., on June 20. Born in 1903, he studied at Chicoutimi and at Laval University where he received his medical degree in 1928. Since that date he had practised in Quebec and was head of the department of orthopædics at the Hôpital de l'Enfant-Jésus. He was also president of the medical council of the same hospital and *professeur agrégé* at Laval University. Dr. Pouliot was a member of the Royal College of Canada, the overseas section of the Société française d'Orthopédie et de Traumatologie, and the International Society of Orthopædics, and was president of the Provincial Association of Orthopædics and Traumatology. In 1944 he became a fellow of the International Academy of Medicine. He is survived by his widow, two daughters and one son.

DR. G. GUSHUE-TAYLOR died on April 23 at the age of 71. He was returning to Canada from Formosa on a freighter when he developed acute abdominal symptoms and died before aid could be had from the patrol vessel which was delayed by stormy seas. Dr. Taylor was born in Bay Roberts, Nfld., and graduated in London, Eng., in 1911, later obtaining his F.R.C.S. He came to Canada around 1920 and early in his career took up medical missionary work in Japan. He finally settled in Formosa where his life work was to be carried out. He was superintendent of the MacKay Memorial Hospital in Formosa. When Dr. Taylor went to Formosa there was no attempt to deal with leprosy in the community. He set to work to collect money in Canada and elsewhere and finally built a treatment centre for leprosy in Formosa himself. Later the Japanese government was so much influenced by his work that it built a leprosarium and gave Dr. Taylor one of its highest honours. A man of intense energy and high resolution, he was of a truly devout mind and always made it clear that the religious aspect of his work was the predominant one. He had an evangelist attached to his hospital who carried on missionary work, and prayers were said in the hospital daily. His wife, a Newfoundland girl, predeceased him; he leaves no family.

The many friends of Mamie Church, the extremely popular wife of Dr. Harcourt B. Church, of Aylmer, P.Q., who was President of the Canadian Medical Association in 1951-52, will learn with the deepest regret of her passing on August 5 in hospital at Ottawa. The Association extends its sympathy to its Past President on this sad occasion.

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ABSTRACTS *from current literature*

MEDICINE

An Outbreak of Trichinosis in Liverpool.

SEMPLE, A. B. *et al.*: BRIT. M. J., 1: 1002, 1954.

An epidemic of 82 cases of trichinosis traceable to pork is described by the authors. Of interest is the fact that five persons gave a history of handling but not eating the suspected sausages: it is suggested that infection arose either via a knife used later to prepare another dish or by the hands. Infection in most cases was attributable to sausages; 34 patients had eaten raw sausages and a further 33 had eaten cooked sausages: the authors suggest that what is regarded as normal cooking may not, in fact destroy the larvæ. Apparently cooks are particularly liable to the disease, the authors noting "time and time again that the only person affected in a household was the housewife." A survey was made of the habits of the housewife and out of 3,092 replies 497 (16.1%) were found to be habitual raw-sausage eaters or tasters.

The symptoms varied considerably from case to case, but swelling of the eyes occurred in all cases and headache was common. Muscular pains, insomnia and splinter hæmorrhages in the nails were also commented on. A somewhat unusual feature of the series was the presence of signs indicating involvement of the central nervous system; one patient died of myocarditis. The authors comment that any suggestion of cardiac involvement in patients with trichinosis should be taken seriously and the patient nursed in a recumbent position.

W. F. T. TATLOW

Gastric Ulcer.

MCCARTHY, F. P. AND MCCARTHY, P. L.: NEW ENGLAND J. MED., 250: 499, 1954.

The authors review the problem of gastric ulcer in the light of a careful study of a group of 138 cases. Allowing for inadequate follow-up in a minimal number the maximal rate of occurrence of carcinoma was approximately 5%. The patient's age and the location of the ulcer were of some diagnostic importance, carcinoma occurring in ulcers on the greater curvature especially, and also being more likely in the older age group. The duration and pattern of symptoms, the amount of weight loss, the degree of acidity on the fasting specimen and the size of the ulcer are not of appreciable significance in differentiating benign from malignant lesions. Rapid and complete healing of a gastric ulcer is acceptable proof of benignity (although not infallible).

The advisability of surgery is an individual problem and requires careful evaluation. In general, prompt surgery is indicated if there are x-ray findings suggesting malignancy, if there is histamine achlorhydria or if the ulcer is on the greater curvature. Surgery is also indicated in good risk patients older than 50 years and in patients under 50 who do not show rapid and complete healing on medical therapy or who have a recurrence.

NORMAN S. SKINNER

The Effect of Methantheline Bromide (Banthine) on Pulmonary Ventilation in Bronchial Asthma and Pulmonary Emphysema.

SJOERDSMA, A. AND DODGE, H. T.: AM. J. M. SC., 227: 255, 1954.

Stimulating the peripheral end of the cut vagus nerve in the experimental animal leads to bronchial constriction; sympathomimetic and parasympathetic blocking agents tend to prevent bronchoconstriction.

Eleven cases of pulmonary emphysema or of bronchial asthma in whom there was limitation of pulmonary ventilation were studied and the effects subjectively, clinically, and in terms of laboratory improvement of giving intravenous Banthine noted. There was worthwhile improvement in eight of the 11, subjectively, clinically and as measured by the vital capacity and maximum breathing capacity. Thickening of the bronchial secretions rendering them more difficult to raise has been noted with Banthine but this did not occur and on the whole, the clinical effects were favourable, even surpassing those found with aminophylline. Tachycardia, dry mouth and blurred vision occurred for three or four hours following administration; one patient with prostatic hypertrophy developed urinary retention. The dose was 50 mgm. of methantheline bromide (Banthine) in 10 c.c. of isotonic saline, given intravenously over a period of one minute.

G. A. COPPING

Antibiotic and Chemotherapeutic Agents in the Treatment of Infantile Diarrhœa and Vomiting.

ALEXANDER, M. B. *et al.*: LANCET, 2: 1163, 1953.

Of great importance is this two-year report released by the Antibiotics Clinical Trials Committee of the Medical Research Council in Great Britain. The efficacy of certain therapeutic agents was studied in 10 different treatment centres and the report covers 1,168 cases, (789 mild, 379 severe): 154 cases were treated with aureomycin; 415 with chloramphenicol; 247 with sulfadiazine. Controls (those treated routinely) numbered 352. All centres agreed on oral administration of the agent employed by them. Dosage used was: aureomycin, 75 mgm.; chloramphenicol, 75 mgm.; and 125 mgm. sulfadiazine per pound of body weight, for a period of seven days.

Clinical progress after start of treatment was based on these criteria; (1) average duration of diarrhœa; (2) average time to full clinical recovery; (3) proportion of mild cases progressing to severe.

Sulfadiazine appeared as the drug of choice for the following reasons: (1) The lowest fatality-rate (1.6%) was in this group. (2) Shortest duration of diarrhœa; (3) Rapid and full clinical recovery. There was a definitely greater chance of toxic manifestations appearing in those treated with chloramphenicol and aureomycin's only value lay in reducing the number of severe cases.

More than 50% of the controls made a straightforward recovery but almost four times as many of this group went from the mild to severe category and the fatality rate (4%) was twice that of the group treated with sulfadiazine. Supportive therapy must not be overlooked.

ISABEL M. LAUDER

SURGERY

Surgical Technique in the Treatment of Duodenal Ulcer by Antroduodenectomy and X-ray Irradiation.

BROWN, G.: BRIT. J. SURG., 41: 359, 1954.

The usual criticisms of the Polya gastrectomy for duodenal ulcers—dumping syndrome, afferent loop syndrome, failure to gain weight—led to a reconsideration of a limited gastrectomy, end-to-end anastomosis and reduction of acid secretion by x-ray. Emphasis is laid on the removal of the ulcer and the maintenance of the normal food route by gastroduodenostomy. Anastomosis of a short duodenal stump is felt to be safer than closure to form a blind stump. The irradiation is given after full recovery from the operation and is usually 2,000 r given over three weeks.

A series of 31 patients with severe chronic duodenal ulcer were subjected to this procedure. One 76 year old man died in 10 weeks of hæmorrhage from an

anastomotic ulcer, and one 72 year old woman had postoperative obstruction relieved by gastroenterostomy. In 29 patients the operation was a success: Convalescence short, weight recovery satisfactory and symptoms relieved. Final evaluation must await a longer period of follow-up. BURNS PLEWES

The Treatment of Difficult Inguinal Herniæ with Tantalum Gauze.

BURNELL, G. F.: BRIT. J. SURG., 41: 354, 1954.

During five years, 47 cases of difficult inguinal hernia in men were repaired with a tantalum gauze implant. All had been done at least 12 months before this report and all were "bad risk" cases from the point of view of recurrence. The principle of replacing the weakened transversalis fascia by fibrous tissue around an inert material is analogous to the use of reinforced concrete. Silver, nylon and silk filigrees provoke too much foreign body reaction, but tantalum is incorporated in the mass of fibrous tissue without inflammatory cells. Steel mesh fractures more easily although it is easier for tying knots.

The technique in using the tantalum gauze implant is described. The unimportant complications that occurred in this series of operations are reported. There were no recurrences in cases in which the technique described was followed. BURNS PLEWES

Temporal Lobe Epilepsy.

PENFIELD, W.: BRIT. J. SURG., 41: 337, 1954.

This is a Hunterian Lecture delivered at the Royal College of Surgeons. Observations and experiments are described to show the pathological anatomy of the brain in epilepsy. Sensory auras, psychical phenomena and the symptomatalogy are discussed and the conclusion reached that the grey matter over the temporal lobe contains "a ganglionic record of human experience." One normal temporal lobe can do the work of both as far as memory and perceptual judgment is concerned. The most frequent pathological change responsible for seizures is incisural sclerosis due to compression of the brain at birth. If medical treatment fails, partial temporal lobectomy offers a 50% chance of cure and a 25% promise of improvement.

Dr. Penfield predicts that second only to the relief of pain, the relief of focal cerebral seizures will some day constitute the most important therapeutic field in the surgery of the nervous system. BURNS PLEWES

Carcinoma of the Floor of the Mouth.

GARDHAM, A. J.: BRIT. J. SURG., 41: 241, 1953.

This University College Hospital series includes 72 patients treated by many different methods—surgical and/or radiological. Since the results of 27% five year survivals is so like other series, it is suggested that this proportion of growths in the floor of the mouth are relatively benign and localized and can be cured by any one of a number of different treatments. But good palliative results are rare for in 54% of cases there was no evidence of benefit from the treatment given. Neither clinical nor microscopical examination of the carcinoma gives a reliable indication of its malignancy.

Of the 20 curable cases, 10 were treated by operation, five by interstitial irradiation alone, two by irradiation and gland operation and three by tele-irradiation. A preference for surgical removal as a result of this experience is defended, but the side-effects of irradiation which cause so much misery may be overcome.

Cases not suitable for surgery are listed: extensive gland involvement with a small primary, wide submucous extension with small ulceration, cases in which the bone of the lower jaw is expanded, soft growths which may be very highly malignant. The size of the growth is not in itself a contraindication to surgery. A case operated upon by Christopher Heath in 1875, in which a large growth involving the tongue and front of the mandible was removed en bloc with 24 years survival, is described. BURNS PLEWES

The ymphatic Circulation in Lymphœdema.

KINMONTH, J. B. AND TAYLOR, G. W.: ANN. SURG., 139: 129, 1954.

Fifteen patients with idiopathic œdema of the lower limbs were studied by lymphangiography of the skin using Patent Blue V, deep lymphangiography at operation, observations of chylous reflux, roentgen ray lymphangiography, microscopy of excised tissues, tissue fluid analysis, and absorption of radioactive marked protein from the limb. Though evidence of large, dilated, incompetent lymphatics was found in the limbs and there was extremely sluggish lymphatic circulation, no anatomical obstruction in lymph vessels was found. The cause of the lymphatic abnormality was not related to infection. Congenital malformation is possible as is the development of a benign new growth (lymphangioma). The most useful operation appeared to be superficial lymphangiectomy but it is a major undertaking. Transplantation of lymphatics by pedicle skin grafts and ligation of deep lymphatics were less successful procedures. BURNS PLEWES

Squamous Cell Anal Carcinoma.

BUXTON, R. W.: ARCH. SURG., 67: 821, 1953.

The anatomy and pathology of epidermoid carcinoma of the anus is reviewed and various methods of treatment compared. The palliation achieved by radium or x-ray therapy in squamous carcinomas in other parts of the body has not been achieved at this site.

It is suggested that proper treatment for cure of squamous carcinoma arising in the anal canal or perianal skin should be: (1) radical resection of the anus, rectum, sigmoid colon, perianal skin, levator ani muscle and pelvic fascia. (2) Wide resection of the posterior vaginal wall and commissure. (3) Bilateral inguinal node dissection 10 to 14 days later. (4) Radical resection of the inguinal and iliac nodes when clinical involvement is apparent and there is no evidence of distant metastases. BURNS PLEWES

OBSTETRICS AND GYNÆCOLOGY

Abruptio Placentæ.

PAGE, E. W., KING, E. B. AND MERRILL, J. A.: OBST. AND GYNEC., 3: 385, 1954.

The most severe grades of abruptio placentæ are accompanied by systemic effects, some of which are potentially lethal. These include a type of shock which is frequently out of proportion to the degree of hypotension, a disseminated fibrin embolism, an *in vivo* defibrination of the blood leading to uncontrollable hæmorrhage, an ischæmia of the renal cortex, and an activation of the fibrinolytic system in the plasma. It is believed that all these events may result from the escape of biologically active material from the separation site into the maternal circulation. The extent to which the latter process proceeds may vary with the hydrostatic pressure within the uterus and the time which elapses between the onset of concealed hæmorrhage and delivery.

The method of grading the severity of abruptio placentæ should reflect the risks to fetal and maternal life.

Three series of abruptio placentæ cases are presented which illustrate the reasons for conservatism in the mild grades, the value of immediate Cæsarean section in reducing fetal mortality in the moderate grades, and the extreme hazards of delay in delivery in the severe grades. Principles of therapy are recommended for each type of abruptio placentæ. ROSS MITCHELL

Fetal Mortality.

HALSEY, H. II: OBST. AND GYNEC., 3: 529, 1954.

Periodic reviews in any institution where obstetrics is done directs attention to one of the paramount remaining problems in the specialty—the fetal mortality rate. This is a measure of the quality of the obstetric care rendered, and such reviews will show where improvement is necessary.

The maximum live-birth survival rate was found in infants weighing 3,250 to 4,000 gm., a fact to keep in mind when doing elective induction of labour and repeat Cæsarean section. Smaller infants, although not necessarily premature, do not do as well.

There is still room for improvement in the fetal mortality rate among the immature infants. Pædiatric assurance of the normal development of these infants would be an added stimulus to further efforts at salvage on the part of the obstetrician. ROSS MITCHELL

Pelvic Tumours other than Fibromas of the Ovary with Ascites and Hydrothorax.

MEIGS, J. V.: OBST. AND GYNEC., 3: 471, 1954.

An arbitrary division has been made between fibrous tumours (fibromas, granulosa cell tumours, thecomas, and Brenner tumours) and epithelial cysts, teratomas and malignant lesions in defining the true Meigs' syndroma. This division is made because there are so many more of the fibrous types of tumours responsible for the syndrome, than the epithelial and teratomous types, which are far more common than the fibromas. The reason for the abdominal fluid is not clear, but the method of chest involvement seems possible of explanation.

It is true that some malignant ovarian tumours may have accompanying ascites and pleural effusion. The chest fluid not only vanishes upon removal of the ovarian neoplasm, but the fluid is not due to metastases, nor does it contain malignant cells that can be recognized by the cytologic method. The importance of the knowledge of chest fluid in pelvic tumours is very great, for some cases considered as hopeless may be curable. ROSS MITCHELL

THERAPEUTICS

Effect of Age on the Utilization of Various Carbohydrates by Man.

ALBANESE, A. A., HIGGONS, R. A., ORTO, L., BELMONT, A. AND DILALLO, R.: METABOLISM, 3: 154, 1954.

Subjects over 40 years of age show a depression in the utilization of glucose, whether given by mouth or by intravenous injection. Differences in the rate of intestinal absorption can, therefore, not explain this phenomenon which occurs with advancing years. It is not associated with a dysfunction of organs concerned with the metabolism of glucose, such as the liver. In contrast, the utilization of fructose by man is depressed only slightly by age. Unlike glucose, fructose is utilized in the formation of glycogen even in the absence of insulin. Fructose also has a greater specific dynamic action than glucose, produces a more rapid rise in the respiratory quotient, is a better ketolytic agent, and may be utilized without transformation to glucose or glycogen by muscles, kidneys, liver, and lungs.

It is concluded from these observations that the pathways of availability of fructose to the body are not affected by age as much as those open to the metabolism of glucose. The practical implication for aged individuals is that the addition of fructose to the diet has a protein-sparing effect, allowing the organism to divert a greater amount of amino acids to protein anabolism than the addition of an equal amount of glucose.

B. L. FRANK

A New Rapid-Action Barbiturate.

WEESE, H. AND KOSS, F. H.: DTSCH. MED. WSCHR., 79: 601, 1954.

A new barbiturate, sodium 5,5-allyl-(2'-methylpropyl)-thiobarbiturate, a yellow alkaline powder readily soluble in water, was used in 10% solution to induce general anæsthesia of short duration in 350 minor surgical interventions in Düsseldorf. The product, known as Baytinal, was first studied by animal experiment. It had the great advantage that the patient was able to go home in 20 to 30 minutes after waking up, though euphoria persisted for one to two hours. The technique was to inject 2 or 3 c.c. of 10% solution rapidly intravenously, wait one to one and a half minutes for loss of consciousness, then slowly inject another 1 to 3 c.c. The maximum dose used was 10 c.c. (1 gm.). Anæsthesia lasted for about 10 minutes. Oxygen inhalation was used as a routine, and succinylcholine injection added when muscular relaxation was needed.

No complications occurred, even in poor-risk cases. Blood pressure remained steady; excitement did not occur. S.S.B.G.

Antihistamine Therapy.

WARIN, R. P.: BRIT. M. J., 1: 1066, 1954.

In this comprehensive article on antihistamine therapy of skin disorders the author points out that the dose of antihistamine required to suppress weals varies greatly from patient to patient. In addition the strength of the stimulus producing the weal may vary; for example additional psychological stress may necessitate an increased dosage of antihistamine. Children are relatively insensitive to antihistamines and consequently a proportionately large dose in relation to age is necessary. Chronic urticaria often fluctuates regularly throughout the 24 hour period, and the antihistamine drugs should therefore be given at the requisite time; by this method one can give relatively large doses of the drug at night for example, avoiding unpleasant side effects of drowsiness.

The author considers all types of antihistamine drugs in relation to treatment. The drug should always be withdrawn slowly because if the tendency to urticaria remains unchanged the patient may find a sudden and full return of the rash very distressing. The main side effect of antihistamine therapy is drowsiness which is commoner with some drugs than with others. Other side effects include dryness of the mouth, headache, blurred vision, nightmares, depression and other mental changes. [The author also describes a symptom which he considers a curious one, but which appears to the abstractor to be typical of the restless feet syndrome.] Some preparations (mepyramine and tripelennamine) give rise to nausea, vomiting, colic and diarrhœa, but these side effects are uncommon if the drug is given after meals. In the author's clinic some patients have received regular treatment with antihistamine drugs for three to four years without ill effect, and the author considers the reported serious effects (such as hæmolytic anæmia, convulsions and jaundice) as of little significance. In conclusion the author points out that the antihistamine drugs benefit only those cases in which itching is part of the eruption. W. F. T. TATLOW

INDUSTRIAL MEDICINE

Advances in the Prevention of Occupational Skin Disease.

BIRMINGHAM, D. J. SR.: AM. INDUST. HYG. A. QUART., 14: 286, 1953.

This is a brief review of some of the advances in prevention of occupational dermatitis which have occurred over the past decade. The author is dealing particularly with personal protective measures. The information is

presented under the following headings: (1) evaluating techniques for preventing occupational and non-occupational dermatitis, (2) educational methods, (3) protective clothing, (4) industrial cleansers, (5) protective ointments.

As more manufacturers employ evaluating techniques before introducing new products, less contact-type dermatitis will be incurred by workmen and consumers. The evaluating techniques in use today are percutaneous (contact) exposure methods as used in lower animals, sensitization studies employing the guinea pig, and the prophetic patch test in human testing. The purpose, the value, and the limitations of each are outlined. Attention is drawn to the fact that results observed in animals cannot always be applied to man although it is often possible to predict the effect. In any ideally conducted industrial hygiene programme, educational methods are important. New employees after examination and proper placement are given detailed instruction regarding the nature of their work, its hazards, and the control methods. Protective clothing of satisfactory material and attractive design is available for different exposures. It has been observed that its use is best controlled in those plants where the clothing is furnished, serviced, and laundered by the plants.

Cleanliness is still the keystone in the programme of prevention and today there is a large selection of industrial cleansers on the market, both liquid and powdered forms and also cleansers of the waterless type. The composition and the uses of the different types are outlined. Reference is made to the introduction of antiseptics, such as hexachlorophene, into industrial cleansers; these are particularly advocated for certain workers such as food handlers and meat packers, and for those exposures where bacteria often play a secondary role in an occupational dermatitis.

Protective ointments or barrier creams may be the best method of prevention in selective cases; but as a general rule these agents are less efficient than other methods of protecting the skin. A more recent development is the use of silicones as barrier agents. The fact that the silicones are inert and capable of repelling water and heavy oils strongly favours their use but as yet it is not known how much laboratory and field evaluation has preceded the introduction of those which are now on the market.

MARGARET H. WILTON

Occupational Diseases with Neurological Symptoms and Signs.

HUNTER, D.: PRACTITIONER, 171: 48, 1953.

In this article the author discusses certain occupational diseases which present neurological symptoms and signs. These include one due to a helminth parasite (cysticercosis), one due to the physical effects of nitrogen bubbles in the tissues (decompression sickness) and others due to the following chemical poisons: lead, mercury, organic mercury compounds, manganese, methyl chloride, tri-ortho-cresyl phosphate, and organic phosphorus insecticides. For each he presents in detail information as to when the cause was first established, the symptoms and signs particularly the neurological, the prognosis, and the known precautions and remedies. The poisonous properties of some of these chemicals have been known since ancient times. That inhalation of lead fumes might cause colic and paralysis was long ago known to physicians in Greece; the Romans were familiar with the dangers of mercury. In the cases of other chemicals knowledge has come more recently. For example, the effect on the nervous system of the dust of manganese compounds has been known in industry since 1922; the poisonous effects of methyl chloride were observed in Chicago in 1929; the first cases of industrial poisoning from tri-ortho-cresyl phosphate in 1944, and, very recently, cases from the use of organic compounds of phosphorus as insecticides.

The present situation in regard to each is indicated and the importance of proper precautions and treatment is stressed. Under controlled conditions the hazard presented is, as a rule, negligible, or of a low order. It is important for a practitioner to be able to find out with any given patient when the symptoms and signs are due to the occupation followed. To ascertain the conditions of work on the spot it is wise for him to visit the factory. Reference is made to legislation in Great Britain to protect the factory worker against accident and disease, and to the effective and progressive work in which the Factory Department is constantly engaged. It is suggested that the practitioner should enlist the help and advice of this Department whenever there is possibility of an occupational factor in diagnosis.

MARGARET H. WILTON

FORTHCOMING MEETINGS

CANADA

INTERNATIONAL CONGRESS OF OPHTHALMOLOGY, Montreal, Que. (Dr. G. Stuart Ramsey, Associate Secretary, Physical Sciences Centre, McGill University, Montreal 2, Que.) September 9-11, 1954.

INDUSTRIAL MEDICAL ASSOCIATION OF THE PROVINCE OF QUEBEC, AND THE INDUSTRIAL SECTION OF THE ONTARIO MEDICAL ASSOCIATION, Joint Meeting, Ottawa, Ont. (Dr. W. F. Prendergast, Secretary of the Section of Industrial Medicine, 22 Commercial Road, Leaside, Toronto 17, Ont.) September 23-25, 1954.

CANADIAN HOSPITAL ASSOCIATION, Biennial Meeting, Ottawa, Ont. (Executive Secretary: Dr. A. L. Swanson, 280 Bloor Street West, Toronto 5, Ont.) May 9-11, 1955.

NINTH INTERNATIONAL CONGRESS ON RHEUMATIC DISEASES, Toronto, Ont. (Dr. Donald C. Graham, Chairman, Committee on General Arrangements, 240 St. George Street, Toronto 5, Ont.) June 23-28, 1957.

UNITED STATES

INTERNATIONAL COLLEGE OF SURGEONS, Convention of the Canadian and American Sections, Chicago, Ill. (Dr. E. N. C. McAmmond, Secretary, Canadian Section, Suite 2, 1701 Broadway West, Vancouver 9, B.C.) September 7-10, 1954.

INTERNATIONAL CONGRESS OF OPHTHALMOLOGY, New York, N.Y. (Dr. William L. Benedict, Secretary-General, 100 First Avenue Building, Rochester, Minn.) September 12-17, 1954.

WORLD CONGRESS OF CARDIOLOGY, Washington, D.C. (Dr. L. W. Gorham, Secretary-General, Second World Congress of Cardiology, 44 East 23rd Street, New York 10, N.Y.) September 12-17, 1954.

INTERNATIONAL ANÆSTHESIA RESEARCH SOCIETY, Annual Congress, Los Angeles, Calif. (Dr. T. H. Seldon, Chairman, 102-110 Second Avenue, S.W., Rochester, Minn.) October 4-7, 1954.

NEW ENGLAND POSTGRADUATE ASSEMBLY, Boston, Mass. (Executive Secretary, R. St.B. Boyd, 22 Fenway, Boston 15, Mass.). October 25-27, 1954.

AMERICAN INSTITUTE OF DENTAL MEDICINE, 11th Annual Meeting, Palm Springs, Calif. (Executive Secretary, Miss Marion G. Lewis, 2240 Channing Way, Berkeley 4, Calif.). October 31-November 4, 1954.

CONGRESS OF NEUROLOGICAL SURGEONS, New York, N.Y. (Dr. Henry M. Suckle, 414 Tenney Bldg., Madison 3, Wisconsin.) November 4-6, 1954.

AMERICAN DERMATOLOGICAL ASSOCIATION, Belleair, Florida. April 17-21, 1955.

AMERICAN PSYCHOSOMATIC Society, Annual Meeting, Atlantic City, N.J. May 4-5, 1955.

OTHER COUNTRIES

THIRD INTERNATIONAL POLIOMYELITIS CONFERENCE, Rome, Italy. (Secretariat: 6 via Lucullo, Rome, Italy. Cable address: Inpolio, Rome.) September 6-10, 1954.

INTERNATIONAL CONGRESS OF INTERNAL MEDICINE, Stockholm, Sweden. (Secretariat of the Third International Congress of International Medicine, Karolinska sjukhuset, Stockholm 60, Sweden) September 15-18, 1954.

CONFERENCE OF THE INTERNATIONAL UNION AGAINST TUBERCULOSIS, Madrid, Spain. (Prof. Alix y Alix, Escuela di Tisiologia, Ciudad Universitaria, Madrid, Spain) September 26-October 2, 1954.

THIRD INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST, Barcelona, Spain. (Secretary General: Prof. Anthony Caralps, Corcega 393, Barcelona, Spain.) October 4-8, 1954.

PAN-PACIFIC SURGICAL ASSOCIATION, Congress, Honolulu, Hawaii. (Dr. F. J. Pinkerton, Director General, Pan-Pacific Surgical Association, Young Building, Honolulu, Hawaii) October 7-8, 1954.

JAPAN MEDICAL CONGRESS, Kyoto, Japan. (Dr. M. Goto, Secretary General, University Hospital, Medical Faculty of Kyoto University, Kyoto, Japan) April 1-5, 1955.

INTERNATIONAL DIABETES FEDERATION, Cambridge, England. (Secretary, P. Duys, 33, Prinsegracht, The Hague.) July 4-8, 1955.

CONGRES DE LA LITHIASE URINAIRE, Evian, (Hte-Savoie), France. (Séc. Prof. Agr. Cl. Laroche, 16, rue Christophe-Colomb, Paris (8e), France) 2-4 septembre, 1955.

THIRD INTERNATIONAL CONGRESS OF VITAMIN "E", Milan, Italy. (Secretary of the Congress, Prof. Emilio Raverdino, Milano, via Pietro Verri 4, Italy.) Early September 1955.

NEWS ITEMS

ALBERTA

We regret to announce the passing of Dr. Lawrence A. Kickham of Westlock, who was drowned near his summer cottage in the Kootney Lake area. Dr. Kickham served overseas with the RCAMC in North West Europe. He was born in Prince Edward Island and graduated from the University of Alberta. He held his specialist's certificate in surgery of the Royal College of Canada.

The Royal Alexandra Hospital in Edmonton is extending its emergency department and making many alterations therein to facilitate the function of the hospital.

To date, poliomyelitis has not raised its horrible head as it did a year ago. The members of the profession are better prepared to combat it this year, however. Preventive vaccination was carried out on a large number of children; it will be interesting to see whether beneficial results are attainable.

W. CARLETON WHITESIDE

BRITISH COLUMBIA

The dog days are upon us, and there is little to record for the past month, now that "the tumult and the shouting" of the C.M.A. meeting has died away. Everyone is on his or her holidays or expects to be. Vancouver is full to overflowing, since the British Empire Games are in full swing under the distinguished patronage of the Duke of Edinburgh and Lord Alexander of Tunis.

Among the competitors in this great meet, are two men whose names are of interest to us as medical men. The first, and the best known, is Dr. Roger Bannister of England, the great miler, who faces John Landy of Australia. It will be a great battle, whoever wins, as both have broken the four-minute barrier, and there is no doubt it will be a close race.

The other name is that of Emmanuel Ifeajuana from Nigeria, a 20-year old lad, whose ambition it is to become a doctor, an ambition which, we understand, will be greatly furthered as a result of this expedition. He has already broken all records in the high jump. He runs with his left foot bare, and a shoe on his right foot. He is a short man, five and a half feet tall, and this makes his achievement the more remarkable.

The captain of the English team is also a medical man, Sir Arthur Porritt, F.R.C.S., a member of Her Majesty's private medical staff.

Dr. Joseph L. Gayton, late chief medical officer in the Victoria-Esquimalt health district, has joined the Metropolitan Health Department of Vancouver lately, as assistant medical health officer.

Dr. Gayton, who was born in New Brunswick, has had a wide experience in public health work. He is a graduate in medicine of the University of Manitoba.

The Delta Municipal Council has approved plans for the building of a new Health Centre in Ladner. This is made possible to a great extent by the generosity of the Kinsmen's Club of Ladner, who are sponsoring the project.

The building will have offices for two public health nurses, and there will be a physiotherapy department.

J. H. MACDERMOT

MANITOBA

The Historical Committee of the Manitoba Pharmaceutical Association, with Professor D. McDougall as chairman and editor, is to be congratulated on the publication of *The History of Pharmacy in Manitoba 1878-1953*. The format is pleasing, the text is well written and the illustrations are interesting. There is considerable medical history, as many of the early doctors in the province conducted a drug store as a supplement to practice. Credit for the first drug store in Manitoba is given to Dr. John Christian Schultz and there is a fine illustration of Apothecaries Hall on Main Street, Winnipeg, owned by Dr. Curtis James Bird. The first soda water fountain in the west was installed in this store in 1873.

Dr. Adam Little of Dauphin has been appointed instructor in clinical medicine at New York University, Bellevue Medical Centre, Postgraduate Medical School.

Construction has begun on an annex which will house St. Boniface Hospital's cobalt therapy unit. The combined service clubs of St. Boniface, Norwood and St. Vital communities are conducting a campaign to raise funds for construction of the building.

Dr. Roper Cadham, Deputy Health Officer of Winnipeg, considers that there will be no serious outbreak of poliomyelitis in the province this year.

Dr. Robert L. Cooke has returned to Winnipeg to assume duties in the Medical College and Winnipeg General Hospital after an absence of three and a half years spent as resident in surgery at the Memorial Centre for Cancer and Allied Diseases, New York City. Dr. Cooke held a McEachern fellowship from the Canadian Cancer Society.

Dr. Arnold Rogers recently addressed the American Society of Gastro-Enterology at its annual meeting in San Francisco. The subject of his address dealt with the care of patients following major abdominal surgery, and was entitled "The Life History of Patients with Ileal Stomas." The address was based on a survey done by Dr. Rogers while a Fellow in Medicine at the Mayo Clinic in Rochester, in conjunction with Drs. B. Marden Black and J. Arnold Bargen, both on the staff of the Mayo Clinic.

Dr. Rogers was an honour graduate of the University of Manitoba, and prior to proceeding to the Mayo Clinic was a research assistant at the Winnipeg General Hospital in the Department of Clinical Research of the University of Manitoba.

Dr. Rogers intends to return to Winnipeg in October when he will assume practice in internal medicine in association with the Mall Medical Group.

Dr. M. K. Kwong, a graduate of the University of Paris, who recently came from Hong Kong, has joined the staff of Clearwater Lake Sanatorium.

Dr. Percy Barsky has opened an office at 1471 Main Street, Winnipeg, for the practice of pædiatrics.

Dr. A. A. Klass has been elected chairman of the Winnipeg men's branch of the Canadian Institute of International Affairs.

Dr. Cecil G. Sheps, formerly of Winnipeg, has been appointed lecturer in preventive medicine in the faculty of Harvard University Medical School.

ROSS MITCHELL

NEW BRUNSWICK

That versatile artist in many media, Dr. P. C. Laporte of Edmundston, has completed a pulpit for Saint John the Baptist Anglican church in Edmundston. Carved in oak in pure gothic style, the pulpit resembles a goblet. The design by Dr. Laporte is beautiful and symbolic and has created much interest. The actual carving was done by Dr. Laporte and some of his senior students and attention is again drawn to the interest in art that has developed in Madawaska due to the skill and enthusiasm of this gifted physician in wood carving and allied arts. What began as a doctor's hobby has developed into an inspiration to many of the young people of a wide area in New Brunswick and Quebec.

Working out of Edmundston, the mobile X-ray Unit of the Tuberculosis Association of New Brunswick, visited pulpwood camps of the Fraser Companies and, on its first forest excursion, 350 employees of this company had chest films recorded. The co-operation of the N.B. Department of Health Services and the company executives made the success of the venture possible. This is the first time in New Brunswick and perhaps in Canada that such a type of examination was made available in forest operation. The Fraser Companies hope that in time all their woods employees will be thus x-rayed.

Dr. Norman Skinner of Saint John, has completed his course in roentgenology at the Peter Bent Brigham Hospital, Boston, and has joined the x-ray staff of the Saint John General Hospital.

Three recent graduates in medicine are doing post-graduate studies in pathology in preparation for appointment to the staff of the N.B. Provincial Pathologist. These courses are possible on bursaries supplied by the federal department of health. Dr. L. Bernier is studying at Laval University, Quebec City. Dr. C. J. Alexander of Fredericton is beginning his three year course at the Peter Bent Brigham Hospital in Boston and Dr. Alfred B. Bastarache of Shediac is working in the Provincial Laboratories in Saint John with the Director of Laboratories, Dr. R. A. H. MacKeen.

Dr. D. F. Sutherland has been promoted to the full rank of obstetrician and gynæcologist in the Saint John General Hospital.

Dr. L. I. Morgan and Dr. W. B. Orser have been appointed to the associate staff of the Saint John General Hospital.

A. S. KIRKLAND

NOVA SCOTIA

Dr. C. B. Stewart, Professor of Epidemiology, Dalhousie University, was appointed Dean of the Faculty of Medicine following the sudden death of Dr. H. G. Grant early in May. Dr. Stewart is a member of the advisory committee on medical research of the National Research Council, the Specialty Board in Public Health of the Royal College of Physicians and Surgeons of Canada, and the Examining Board of the Medical Council of Canada and is associated with many of the medical and public health associations in Canada and the United States, holding executive positions in several. During the war Dr. Stewart served with the Royal Canadian Air Force in the rank of Wing Commander, as commanding officer of medical research units responsible for studies of the medical problems of high altitude flying. Recently he had the honour of being invited to participate in the deliberations of a committee to review the future programme of research and teaching in epidemiology at Johns Hopkins University. His status in medical research was also recognized in his appointment, in 1948, as chairman of a committee to survey medical research facilities in Canada for the National Research Council, Defence Research Board and the Department of National Health and Welfare.

The Sydney Polio Clinic which is to be the emergency poliomyelitis clinic for Cape Breton was opened in June. The doctors in charge of the clinic and for whom special training was provided by the Nova Scotia Department of Health are Dr. Arthur Ormiston and Dr. C. A. D'Intino. The Nova Scotia chapter of the Poliomyelitis Foundation has donated $2,400.00 worth of equipment to the clinic and will lend its support in every possible way.

Dr. Peter S. Campbell of Halifax, former Deputy Minister of Health and Welfare for Nova Scotia, and dean of Canadian Public Health Officers, was honoured by the Canadian Tuberculosis Association with life membership at their annual meeting in Saint John. Dr. Campbell entered the health department as the first provincial tuberculosis officer in Nova Scotia and in due course

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was appointed Deputy Minister of the Department of Health. The high standard of the tuberculosis control programme in Nova Scotia is in no small measure due to his efforts and to his ability to choose men of outstanding ability to carry on the work of the department.

Dr. A. A. Giffin of the Kentville School Board has been named vice-president of the first meeting of the Nova Scotia Association of Urban and Municipal School Boards. Dr. Giffin was a Surgeon-Captain in the Royal Canadian Navy during the war years and is now a specialist in internal medicine in the Valley town.

Dr. H. B. Ross of Halifax was invited through the courtesy of the Lederle Laboratories of New York to attend a one day postgraduate course of instruction given by the Regina and District Medical Society at Regina in May. The purpose of Dr. Ross's visit was to make a study of this meeting, because Lederle Laboratories plan to underwrite a similar course in Nova Scotia under the auspices of the Postgraduate Committee of the Dalhousie Medical School.

Federal aid, totalling $14,400, in operating a Mental Health Clinic for Children at Halifax has been announced by the Department of National Health and Welfare. Another grant of $3,000 to the Nova Scotia Sanatorium at Kentville was announced at the same time.

Provision of modern quarters for a Provincial Polio Clinic at Halifax is being assisted by a federal health grant, announced by the Department of National Health and Welfare. This is part of a provincial programme involving also a modernly-equipped branch clinic at Sydney. The new facilities at Halifax have been made possible through remodelling of a brick building on grounds of the old Victoria General Hospital.

Dr. W. D. Stevenson of Halifax, Dalhousie Medical School, recently returned from attending a meeting of the Harvey Cushing Society in Sante Fe, New Mexico.

The *Dalhousie Medical Journal* which had a brief period of success between 1936 and 1939, only to be abandoned with the outbreak of war, has been revived under the sponsorship of the Dalhousie Medical Students' Society. The first issue of the new series lists three principal objectives: (1) to encourage the Dalhousie undergraduates in scientific writing; (2) to encourage the habit of reading medical material other than the specified textbooks; (3) to increase the interest of practising physicians in research at Dalhousie by publishing accounts of projects going forward.

Honorary editor during the year was Irving H. Koven, who has been active in promoting the journal's revival. Others on the staff are: Henry J. Presutti, Editor in chief; W. C. Elliott, P. B. Jardine, and D. T. Janigan, associate editors; Jack L. Fairweather, managing editor; A. I. Lesser, circulation manager; M. Aronoff, advertising manager; W. S. Huestis, D. Saffron, and P. B. Black, editorial assistants.

Faculty advisers are Professors C. B. Stewart, J. G. Aldous, and R. L. Saunders. C. M. HARLOW

ONTARIO

The Niagara Falls Medical Society is holding a Centennial Clinical Day on Saturday, September 18, 1954. There will be speakers from the Mayo and Crile Clinics.

The United States National Foundation for Infantile Paralysis has made a March of Dimes grant of $179,000 to the Connaught Medical Research Laboratories to improve methods for growing poliomyelitis virus in the large quantities needed for vaccine production. Connaught research workers have contributed a great deal to large scale preparation of poliomyelitis virus culture fluids. They delivered more than 380 gallons of this fluid between September 1953 and February 1954; this was used as the starting point for the manufacture of poliomyelitis vaccine now being tested in the United States. Quantity production of the virus was achieved by growing it on tiny fragments of minced monkey tissues nourished in a fluid culture medium developed at Connaught Laboratories. The mixture contains 63 ingredients.

Dr. F. Van Nostrand, at present director of psychiatric and neurological treatment service, Sunnybrook, Toronto, has been appointed director of a new psychiatric and neurological treatment service for the inmates of Ontario prisons. A special treatment ward for psychopaths will be established at the new maximum security prison being constructed at Millbrook. Treatment will be provided for drug addicts at the Mercer Reformatory for women and at the Mimico Reformatory for men. Other psychiatric problems involved in handling a daily prison population which averages about 5,000 will be studied.

Nine hospitals have received construction grants totalling one million dollars from the provincial government. Women's College Hospital heads the list with $560,333. Scarboro General Hospital received $197,000. Other hospitals which obtained grants are: Lady Minto Hospital, Cochrane; Perley Home for Incurables, Ottawa; Hotel Dieu, Kingston; St. Catharines General Hospital; Salvation Army Grace Hospital, Windsor; Parkwood Hospital for Incurables, London and St. Mary's Memorial Hospital, St. Mary's.

The Ontario government has set aside more than $350,000 for 16 hospitals throughout the province in connection with special grants of $1,000 for each 300 square feet for organized out-patient departments and for auxiliary services accommodation. These grants will be released to the hospitals as soon as matching grants have been approved by the Federal Government.

Dr. Stanley Hartroft, Professor, Banting and Best Department of Medical Research, has been appointed professor of pathology at Washington University, St. Louis, Missouri.

Dr. R. I. Harris has been named by the Royal College of Surgeons as Sims Commonwealth Travelling Professor for 1955. He is the first Canadian to be so honoured. Dr. Harris will lecture throughout Australia and New Zealand. LILLIAN A. CHASE

SASKATCHEWAN

The Saskatchewan Hospital Association is presently proceeding with plans to recruit nurses from Britain to fill an estimated need of 300 vacancies in this province. Advertisements have been mailed to England and a central office set up there to receive the applications. In August, Miss Lola Wilson, registrar of the Saskatchewan Registered Nurses' Association, left for England to interview and screen the applicants. In this way it is hoped only fully qualified nurses will be accepted who will be eligible for immediate registration upon the completion of a short orientation period in one of our larger schools of nursing; about three weeks of orientation is felt to be advisable so that the new registrants may become familiar with our drugs and nursing procedures.

During June of this year a new type of job training workshop and work conditioning and testing centre was opened in Saskatoon. This workshop is similar to the one now operated in Moose Jaw, and helps fill a long felt gap in the rehabilitation services offered to the province's handicapped citizens. Operated by the Handicapped Civilian Association, the new workshop will pro-

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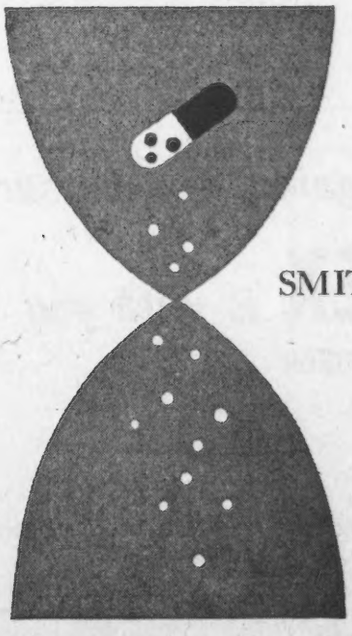

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vide testing and guidance to determine the aptitudes and abilities of handicapped persons for a suitable type of work, conditioning to provide the ability to work on normal work days, and training in a vocation. The establishment of the new workshop has been made possible through the Saskatoon Cosmopolitan Club which has donated $200 so far towards the purchase of equipment and machinery. The operation of the workshop will be made possible through the March of Dimes funds.

Bi-monthly treatment clinics financed by the Saskatchewan Council for Crippled Children and Adults have been started at Melfort to serve that area, with the programme getting under way during May. Sixty-one patients were reviewed by the two directors of the Saskatoon and Regina Physical Restoration Centres, Dr. A. E. Buckwold and Dr. T. J. Roulston, assisted by therapists, social workers and a psychologist.

Under date of June 26, 1954, it has been indicated that no cases of polio have been reported in Saskatchewan for the past month. To date this year there have been 22 cases and one death attributed to the disease.

The Saskatoon City Hospital has recently announced an operating deficit of $66,867 for the first five months of this year. The Hospital Rate Board of the Saskatchewan Hospital Services Plan has approved a request for $20,000 in advance.

Hon. T. J. Bentley, Minister of Public Health, announced that hospitalization tax rates for 1955 will be the same as those in effect for this year. They are as follows: For each self-supporting person or a spouse $15, (including a widowed, divorced, or separated person); for every person who reaches the age of 18 years before January 1, 1955, $15; for each dependent child under 18 years, $5; the maximum tax for a taxpayer, his spouse, dependents under 18 years, children over 18 who are incapacitated by reason of physical or mental infirmity and dependents 18 years or over but under 21 years on January 1, 1955 who are attending educational institutions or training at a school of nursing, $40.

G. W. PEACOCK

NEWS OF THE MEDICAL SERVICES

Canadian Armed Forces

Brigadier K. A. Hunter, O.B.E., C.D., Q.H.P., R.C.A.M.C., Director General of Medical Services, presented a paper entitled "The R.C.A.M.C. in the Korean War" to the Armed Forces Section of the Canadian Medical Association annual conference in Vancouver. Col. J. E. Andrew, E.D., R.C.A.M.C., of the staff of the Director General Medical Services was co-author of this paper.

Major J. F. Evans, R.C.A.M.C. commenced a year's postgraduate study in psychiatry on July 1, 1954 at the Walter Reed Army Medical Centre, Washington, D.C.

Lt. Col. J. H. Watson, Lt. Col. J. R. Feindel, and Major W. B. Hogarth, C.D., R.C.A.M.C., recently were granted the Diploma of Public Health by the University of Toronto.

Capt. J. J. Glynn and Capt J. K. O'Shaughnessy were selected to attend the forthcoming course for the Diploma of Public Health at the University of Toronto.

Lt. Col. R. A. Smillie, M.B.E., is attending a Senior Officers Course at the Royal Army Medical College, Millbank, U.K.

Lt. Col. B. L. P. Brosseau, O.B.E., M.C., C.D., has been appointed Senior Medical Officer Canadian Army Liaison Establishment, London, England, replacing Lt. Col. P. A. Costin, who returns to Canada to become the Area Medical Officer, Eastern Ontario Area, Kingston, Ontario.

Major J. E. Gilbert recently completed a residency in Psychiatry at Queen Mary Veterans' Hospital, Montreal, and has been posted as assistant to AMD 8, the professional consultant of the Director General Medical Services staff.

Group Captain D. G. M. Nelson, former Deputy Director of Medical Services R.C.A.F. has been appointed Commanding Officer of the Institute of Aviation Medicine, Toronto, effective October 1, 1954 replacing Group Captain B. C. Coles who is commencing postgraduate training at the University of Toronto. Group Captain B. R. Brown has completed postgraduate training in Industrial Hygiene at Harvard University and has been appointed Deputy Director of Medical Services, R.C.A.F., effective October 1, 1954.

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NEWS AND NOTES

NATIONWIDE TELEVISION IN POSTGRADUATE MEDICINE

On Thursday evening, September 23, 1954, from 6.00 p.m. to 7.00 p.m., Eastern Daylight Saving Time, the American College of Physicians will utilize television through a national closed circuit over the Columbia Broadcasting System to carry to its members and their colleagues a Symposium on the Management of Hypertension. This telecast is made possible through the cooperation and generous support of Wyeth Incorporated of Philadelphia, and will be the *first nationwide* closed circuit hookup for postgraduate medical education.

The panel of distinguished physicians who will participate includes: Dr. Cyrus C. Sturgis, President, American College of Physicians; Dr. F. H. Smirk, Professor of Medicine, University of Otago, New Zealand; Dr. R. W. Wilkins, Chief, Hypertension Clinic, Massachusetts Memorial Hospital, Boston; Dr. Garfield G. Duncan, Director of the Medical Division, Pennsylvania Hospital, Philadelphia; and Dr. Edward D. Freis, Adjunct Clinical Professor of Medicine, Georgetown University, Washington.

A "closed TV circuit" is one by which reception is controlled and not open to the general TV public. This telecast cannot be picked up in the home, but the invited audience must go to the TV receiving station. Twenty-three such receiving stations will be used; these will be located in Boston, New York, Philadelphia, Washington, Pittsburgh, Charlotte, Atlanta, Cincinnati, Detroit, Chicago, St. Louis, Milwaukee, Minneapolis, Memphis, Dallas, Houston, New Orleans, Denver, Salt Lake City, Los Angeles, San Francisco, Baltimore and Cleveland.

MEDICAL RESEARCH FELLOWSHIPS

The Division of Medical Sciences of the National Academy of Sciences–National Research Council, is accepting applications for postdoctoral research fellowships for 1955-1956. These awards are designed to offer research experience for promising individuals who look forward to investigative careers, and not to provide practical experience in the clinical field. Ordinarily Fellowships are not granted to persons over 35 years of age. The programme includes:

Fellowships in the Medical Sciences, supported by The Rockefeller Foundation, are administered by the Medical Fellowship Board of the Division. Fellows are expected

(Continued on page 71 of the advertising section)

BOOK REVIEWS

GERIATRIC MEDICINE

Edited by E. J. Stieglitz, Consulting Internist, Suburban Hospital, Bethesda, Maryland and Washington Home for Incurables; Chairman of Staff 1945-1947, Suburban Hospital, Bethesda, Maryland. 718 pp. 3rd ed. illust. $15.00. J. B. Lippincott Company, Montreal, 1954.

This new edition of Dr. Stieglitz's popular text brings the subject matter up-to-date and incorporates two new chapters, dealing with geriatric nursing and the mental hygiene of later maturity, the latter written by the senior author. Geriatric medicine cuts right across the various specialties of medicine and an adequate presentation of so large a field, therefore, requires the knowledge and special experience of many authorities. It is difficult to conceive of any one physician who would be competent to cover all the many aspects of geriatric medicine. The formidable list of contributors gives the reader confidence that the subject could have hardly been dealt with by a more competent team of workers.

Clinical geriatrics is not only concerned with the aged but, even more so, with the process of aging and the prevention of premature senile infirmities. In this edition, valuable material has been added concerning the care and the guidance of normal aging people and the anticipation of disorders liable to develop with advancing years, their prevention and early correction. Material previously included and not directly pertinent to geriatric medicine has now been omitted.

The medical reader of practically every shade of interest cannot fail to find a source of useful information in this book, which covers such a vast variety of subjects connected with geriatric medicine.

EMOTIONAL FACTORS IN SKIN DISEASE

E. Wittkower and B. Russell. 214 pp. $4.00. Paul B. Hoeber, Inc., New York, 1953.

This volume is the joint effort of two outstanding authorities in the fields of psychiatry and dermatology. The commending forewords of Dr. R. M. B. MacKenna and Dr. D. Ewen Cameron attest to its sterling worth.

Dr. Wittkower not only writes with authority of his own specialty, but also exhibits in the discussion of dermatoses a familiarity with skin conditions rarely demonstrated by psychiatrists. This dermatological background stems from a long association in practice with Dr. MacKenna and also with his co-author, Dr. Brian Russell. Dr. Russell, from his extensive experience in the London hospitals in both dermatology and applied psychiatry, tempers the psychiatric values which have been written into the dermatological equations.

The book is a lucid exposé of the basic problems of psychiatry which impinge on dermatology. It is based on Paul's premise that mind and body, as parts of the individual, make the whole man, who reacts as one unit to factors of stress or of disease. The authors make a plea to the profession to explore further and understand the hitherto unrecognized psychological factors in disease states, and to attempt to correlate these symptoms and findings with the presenting skin pathology.

The book's orderly format is to be commended. Starting from the development of the skin, the chapters proceed to a description of its functions, and then to a discussion of neuroanatomy and physiology, in an effort to supply the basic data so necessary for a complete understanding of the emotional factors in skin disease. The writers' general conclusions are backed up by a wealth of material collected from controlled group studies, concise case histories, and a carefully indexed bibliography.

The chapter on rosacea, written in collaboration with the late Dr. R. Klaber, is especially illuminating and high-lights the general tone of the monograph. Dermatologists will have difficulty in following Dr. Peter

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Edgehill in his writings on "eczema". This writer has attempted the Herculean task of classifying multiple dermatoses under this one heading and has drawn his conclusions from these diversified entities. The intriguingly-written chapter by Dr. D. Irwin cleverly sums up our present-day knowledge of the alopecias. Dr. J. Slorach, in a concise way, presents figures for the incidence of dermatoses in mental patients as compared with those obtained in a skin outpatient department population. He concludes with a general summary of psychotic states and their associated skin conditions.

These authors have pioneered and ventured far afield in the realm of psychomatic medicine and have produced a basic reference book to guide the interested into these relatively untrodden paths. There is much which the morphological dermatologist may glean from this work, as it confirms and explains many impressions held by the profession over the years. It is a fundamental contribution to psychosomatic medicine and will prove to be of untold worth to psychiatrists, dermatologists and to medicine at large.

THE MANAGEMENT OF PAIN

J. J. Bonica, Director, Department of Anæsthesia, Tacoma General and Pierce County Hospitals; Clinical Associate, Department of Anatomy, University of Washington Medical School, Seattle, Washington. 1533 pp. illust. $20.00. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Ltd., Toronto 2, 1953.

This must not be regarded as just another book on this subject but rather as a book of reference which should be on the shelves of every library and consulted by every one who deals with the management of pain.

The introduction deals with man's efforts through the ages to alleviate pain. Following the introduction the contents of the book are itemized chapter by chapter in a clear and concise form which facilitates quick reference points. Special emphasis is placed on the control of pain due to carcinoma and management is outlined according to site of the tumour.

Each chapter is followed by an excellent list of references which round out a section which is complete in itself.

Dr. Bonica is to be congratulated on preparing this great work. The subject has been completely covered and in a manner that makes for easy and pleasant reading.

PARSONS' DISEASES OF THE EYE

Sir Stewart Duke-Elder, Surgeon-Oculist to the Queen; Director of Research Institute of Ophthalmology, University of London. 606 pp. illust. 12th ed. $8.50. J. & A. Churchill Ltd., London, W.1.; British Book Service (Canada) Ltd., Toronto 6, 1954.

When an old favourite has had a 47-year career as a standard text and run through 12 editions, the potential reader's concern is to know whether the outmoded has been eliminated and the new incorporated with the passage of time. The name of Sir Stewart Duke-Elder is sufficient to warrant the expectation that the latest edition of this standard British textbook of ophthalmology will be hale and hearty. Examination of the book confirms this expectation. The new chapter on general therapeutics contains a lucid exposition of the value of antibiotics, cortisone and ACTH, and even Filatov's tissue therapy. There are many changes throughout the text and in the illustrations, and the book can be recommended as a reliable guide to ophthalmology for students and general practitioners.

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AN INTRODUCTION TO PATHOLOGY AND BACTERIOLOGY

E. C. Smith, Sometime Senior Pathologist, Medical Laboratory Service, Nigeria. Revised by R. Kirk, Professor of Pathology in the Kitchener School of Medicine, University College of Khartoum. 390 pp. illust. 2nd ed. 50/-. $8.50. Staples Press Limited, London; British Book Service (Canada) Ltd., Toronto 6, 1953.

Although this introduction to pathology and bacteriology is primarily intended for use by students in the tropics, it could serve for learning the rudiments of the subjects elsewhere, since all the common conditions encountered in temperate climates are discussed. Dr. Kirk's version of this textbook is up to date and clearly written. The chapters on elementary bacteriology are particularly commendable; the chapter on general disturbances of metabolism and circulation rather suffers from lack of illustrations of a notoriously dull subject for students.

PEPTIC ULCER

C. F. W. Illingworth, Regius Professor of Surgery, University of Glasgow, Surgeon, Western Infirmary, Glasgow. 287 pp. illust. $7.15. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Ltd., Toronto 2, 1953.

This monograph on peptic ulcer deals with the problem in all its aspects. The basic physiology, anatomy and pathology are well covered. Fractional test meals as a diagnostic feature have been abandoned, the only useful diagnostic aid from gastric analysis being the presence or absence of histamine-fast achlorhydria. Experimental production of peptic ulcer in animals is well reviewed. The psychosomatic approach and the clinical aspects and diagnosis are thoroughly discussed. Gastroscopy in the hands of an experienced observer is felt to be the most accurate method of deciding whether an ulcer is malignant. A chapter is devoted to medical treatment. The feeling in the literature is that once started, a peptic ulcer tends to persist throughout life and present methods of treatment do nothing more than assist in inducing a remission.

As would be expected, the surgical aspects of peptic ulcer are thoroughly discussed. Except for emergencies such as perforation, etc. the author rarely operates on duodenal ulcers of less than five years' duration. Gastrojejunostomy has a place in the poor-risk patient with duodenal ulcer. It also has a place in women with duodenal ulcer because they have little tendency to develop stomal ulcer after gastro-jejunostomy, whereas they are very liable to develop severe anæmia and nutritional disturbances after gastrectomy. He is conservative in his approach to bleeding ulcer, relying primarily on medical treatment, the most important indications for operation being continuance or recurrence of hæmorrhage. Although nothing new has been presented, the author has put down in a not too lengthy and well-written monograph the pertinent facts about peptic ulcer, which internist and surgeon alike could read profitably.

PHARMACOLOGY

J. H. Gaddum, Professor of Pharmacology in the University of Edinburgh. 562 pp. illust. 4th ed. $5.25. Oxford University Press, Toronto, 1953.

The new edition of this standard medium-sized British textbook merits a repetition of the praise given five years ago to its forerunner (*Canad. M. A. J.*, 60: 546, 1949). It covers the whole field of pharmacology succinctly and clearly. The last chapter in which the author discusses general matters, such as the absorption and fate of drugs, combined effects of drugs, biological assay, toxicity tests, and clinical trials of drugs, is particularly valuable.

THE UNCOMMON HEART DISEASES

N. E. Reich, Clinical Assistant Professor of Medicine State University of New York, College of Medicine. Associate Attending Physician, Kings County Hospital. 516 pp. illust. $11.50. Charles C. Thomas, Springfield, Illinois; The Ryerson Press, Toronto, 1954.

Deaths due to heart disease have risen from fourth to first position in the past half century; small wonder, therefore, that the number of textbooks on heart disease has increased considerably. Nevertheless, the book under review fills a gap and supplements the standard texts on the heart. It covers interesting and unusual conditions in an authoritative manner based on years of careful and painstaking study.

The subject is approached etiologically, since the author believes that understanding of the causative factors should guide prognosis and treatment. Only in conditions in which the etiology is unknown are anatomical changes substituted in the classification. The first chapter on non-structural cardiovascular abnormalities deals with the effects of various conditions of stress. This is followed by chapters on trauma of the heart; tumours of the heart; the so-called "diffuse collagen diseases"; acute nonspecific myocarditis; acute nonspecific pericarditis; chronic constrictive pericarditis; syphilis of the myocardium and valves; tuberculous myocarditis; mycotic infections of the heart; cardiac diseases of parasitic origin; cor pulmonale; nutritional disturbances; vitamins and heart disease; effects of metabolic disorders; the heart in blood dyscrasias; the association of neuromuscular diseases and cardiopathies; effects of drugs, electrolytes, and toxins; non-arteriosclerotic diseases of the coronary arteries; and various diseases of the myocardium, pericardium and the venous system.

The book is well written and well illustrated. Controversial matters are presented in an impartial way and obsolete views, which have been handed down from generation to generation of physicians, have been entirely excluded. The subject is presented to the reader in a way which makes it easy for him to commit the facts to memory.

Books Received

Books are acknowledged as received, but in some cases reviews will also be made in later issues.

Expert Committee on Rheumatic Diseases. First Report. 25 pp. 1/9d; $0.25. World Health Organization, Palais des Nations, Geneva, April 1954.

Expert Committee on Environmental Sanitation. Third Report. 25 pp. 1/9d; $0.25. World Health Organization, Palais Des Nations, Geneva, April 1954.

An Introduction to Pathology and Bacteriology. E. C. Smith, Sometime Senior Pathologist, Medical Laboratory Service, Nigeria. Revised by R. Kirk, Professor of Pathology in the Kitchener School of Medicine, University College of Khartoum. 390 pp. Illust. 2nd Ed. 50/-. $8.50. Staples Press Limited, London; British Book Service (Canada) Ltd., Toronto 6, 1953.

Hypertension and Nephritis. A. M. Fishberg, Director of Medicine, Beth Israel Hospital, New York; Clinical Professor of Medicine, Post-Graduate Medical School of New York University, New York. 986 pp. Illust. 5th ed. $12.50. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Limited, Toronto, 1954.

The Eczemas. Edited by L. J. A. Loewenthal, Lecturer in Dermatology, University of Witwatersrand; Dermatologist, General Hospital, Johannesberg. 267 pp. Illust. $6.00. E. & S. Livingstone, Ltd., Edinburgh; The Macmillan Company of Canada Limited, Toronto, 1954.

Roentgenographic Technique. D. A. Rhinehart, Emeritus Professor of Anatomy and Roentgenology, Univerity of Arkansas; Honorary Member American Society of X-ray Technicians. 454 pp. Illust. 4th ed. Revised. $8.50. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Limited, Toronto, 1954.

Annual Review of Medicine. W. C. Cutting and H. W. Newman, Editor and Associate Editor, Stanford University School of Medicine. 490 pp. Vol. 5. $7.00. Annual Reviews, Inc. Stanford, California, 1954.

Physiology in Diseases of the Heart and Lungs. M. D. Altschule, Assistant Professor of Medicine, Harvard Medical School; Visiting Physician and Research Associate, Beth Israel Hospital. 554 pp. Revised ed. $7.50. Harvard University Press, Cambridge, Mass., 1954.

Alpha Tocopherol (Vitamin E) in Cardiovascular Disease. W. E. Shute and E. V. Shute. 238 pp. Illust. $7.00. The Shute Foundation for Medical Research, London; The Ryerson Press, Toronto, 1954.

The Vitamins in Medicine. F. Bicknell, Honorary Physician, French Hospital, London, and F. Prescott, Clinical Research Director, The Wellcome Foundation, London. 784 pp. Illust. 3rd ed. Revised. 70/-. $12.00. William Heinemann Medical Books Ltd., London; British Book Service (Canada) Ltd., Toronto 6, 1953.

The Public Health Inspector's Handbook. H. H. Clay, Fellow of the Sanitary Inspectors' Association. Major, R.A.M.C.—Late of the Army School of Hygiene. 608 pp. Illust. 8th ed. Revised. 30/-. H. K. Lewis & Co., Ltd., London, 1954.

Textbook of the Nervous System. H. C. Elliott, Associate Professor of Anatomy, College of Medicine, University of Nebraska. 437 pp. Illust. 2nd Ed. $9.00. J. B. Lippincott Company, Montreal, 1954.

Infant Feeding and Feeding Difficulties. P. R. Evans, Director, Department of Child Health and Children's Physician, Guy's Hospital; and R. MacKeith, Assistant Children's Physician, Guy's Hospital. 277 pp. Illust. 2nd Ed. $2.20. J. & A. Churchill Ltd., London, W.1.; British Book Service (Canada) Ltd., Toronto 6, 1954.

Auditory Disorders in Children. H. R. Myklebust, Professor of Audiology, School of Speech and Director, Children's Hearing and Aphasia Clinic Northwestern University. 366 pp. $6.50. Grune & Stratton, New York; The Ryerson Press, Toronto, 1954.

Pediatric Problems in Clinical Practice. H. Michal-Smith, Chief Clinical Psychologist, Flower and Fifth Avenue Hospitals; Research Associate in Pædiatrics, New York Medical College, New York City. 310 pp. $6.00. Grune & Stratton, New York; The Ryerson Press, Toronto, 1954.

Digital Plethysmography. G. E. Burch, Henderson Professor of Medicine, Tulane University School of Medicine. 134 pp. Illust. $5.50. Grune & Stratton, New York; The Ryerson Press, Toronto, 1954.

Diseases of the Liver. M. A. Spellberg, Associate Professor of Clinical Medicine, University of Illinois School of Medicine. Associate Attending Physician, Department of Medicine, Michael Reese Hospital, Chicago, Illinois. 646 pp. Illust. $18.25. Grune & Stratton, New York; The Ryerson Press, Toronto, 1954.

Annual Review of Medicine. W. C. Cutting, and H. W. Newman, Editor and Associate Editor, Stanford University School of Medicine. 490 pp. Vol. 5. $7.00. Annual Reviews, Inc., Stanford, California, U.S.A., 1954.

A Primer of Pulmonary Function. H. G. Trimble, and J. Kieran. 22 pp. California Tuberculosis and Health Association.

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JOURNAL OF

Canadian Medical Association

Editorial offices — 3640 University St., Montreal 2
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Contributors: Articles are accepted on condition that they are contributed solely to this Journal. Material contributed to this Journal is covered by copyright, and permission must be obtained for its reproduction either in part or in whole.

Manuscripts must be typewritten, double spaced, and the original copy.

Papers should be kept below 4,000 words wherever possible. Whilst not necessarily a cause for rejection, excessive length of an article is undesirable.

References: in the case of a journal arrange as follows: author (JONES, A. B.), title, journal, volume, page, year. In the case of a book: WILSON, A., Practice of Medicine, Macmillan, London, 1st ed., p. 120, 1922.

Illustrations: A limited number will be accepted. Photographs should be clear: drawings should be in india ink on white paper. All unmounted. Legends to be typed separately.

Reprints: May be ordered upon forms sent with galley proofs.

News: The Editor will be glad to consider any items of news that may be sent in by readers.

CLASSIFIED ADVERTISEMENTS

Send copy to Canadian Medical Association, 3640 University Street, Montreal, not later than the fifteenth of the month previous to issue.

Rates: $3.00 for each insertion of 40 words or less, additional words 5c each.

If a box number is requested, there will be an additional charge of 50c on the first advertisement to cover postage and handling charges.

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CLASSIFIED ADVERTISEMENTS

POSITION VACANT.—Certified obstetrician and gynæcologist required by medical clinic in southern Ontario to assist in the practice of obstetrics and gynæcology. Please give particulars of qualifications, experience, etc., in first letter. Apply Box 952, Canadian Medical Association Journal, 3640 University Street, Montreal, Que.

POSITION VACANT.—Doctor required by group in Northwestern Ontario to engage in general practice and also perform anæsthetist duties. Would prefer at least one year's anæsthetic training. Please give full details regarding age, marital status, training, experience, availability, etc. Reply to Box 983, Canadian Medical Association Journal, 3640 University Street, Montreal, Que.

POSITION VACANT.—Assistant wanted for busy general practice in Toronto. Good salary plus commission. State qualifications, age and nationality. Apply Box 986, Canadian Medical Association Journal, 3640 University Street, Montreal, Que.

POSITION VACANT.—Assistant wanted at once for group doing large general and industrial practice in suburban Toronto. Located in growing community. Modern well equipped clinic building. Good salary to start. Please give full particulars in reply to Box 993, Canadian Medical Association Journal, 3640 University Street, Montreal, Que.

POSITION VACANT.—Wanted. Assistant in two man practice in Alberta mining district. Postgraduate obstetric, surgical and anæsthetic experience desirable. Modern 10-bed cottage hospital controlled by practice. Good unfurnished house available. Salary $500 - $700 monthly dependent on experience. For details write Box 996, Canadian Medical Association Journal, 3640 University Street, Montreal, Que.

POSITION VACANT.—Beginning September 1, assistant, general practitioner to work with two others in town of 3,000, Northern Ontario. Contract basis, position permanent if mutually satisfactory. Modern hospital and office facilities. Offering beginning salary $550 per month. In replying state marital status and religion. Reply to Box 982, Canadian Medical Association Journal, 3640 University Street, Montreal, Que.

Books Received

Expert Committee on Alcohol. First Report. 16 pp. $0.25. World Health Organization, Geneva, 1954.

Expert Committee on Poliomyelitis. First Report. 69 pp. $0.50. World Health Organization, Geneva, 1954.

Directory of International Scientific Organizations. 312 pp. 2nd Ed. $2.50. UNESCO; University of Toronto Press, Toronto, 1953.

Good General Practice. S. Taylor. 604 pp. Illust. $2.00. Oxford University Press, Toronto, 1954.

Clinical Medicine in General Practice. Edited by J. Fry. 436 pp. $4.70. J. & A. Churchill Ltd., London; British Book Service (Canada) Ltd., Toronto 6, 1954.

Practical Child Psychotherapy. C. Boenheim. 184 pp. 2nd Ed. $2.60. Staples Press Limited, London; British Book Service (Canada) Ltd., Toronto 6, 1953.

Practical Clinical Biochemistry. H. Varley, Biochemist, Manchester Royal Infirmary; Lecturer in Clinical Pathology, Manchester University. 551 pp. Illust. $7.25. William Heinemann Medical Books Ltd., London; British Book Service (Canada) Ltd., Toronto 6, 1954.

Ciba Foundation, Colloquoia on Endocrinology. G. E. W. Wolstenholme, General Editor for the Ciba Foundation. Vol. 6. 350 pp. Illust. $6.00. J. & A. Churchill Ltd., London, W.1.; British Book Service (Canada) Ltd., Toronto 6, 1953.

Acta Radiologica Suppl. 108: Temporary Unilateral Occlusion of the Pulmonary Artery. B. Nordenstrom. 148 pp. Illust. Sw. Kr. 30. Acta Radiologica, Stockholm 2, Sweden, 1954.

Acta Radiologica Suppl. 107: The Central Veins and Deep Dural Sinuses of the Brain. C. Johanson. 184 pp. Illust. Sw. Kr. 25. Acta Radiologica, Stockholm 2, Sweden, 1954.

Advances in the Control of Zoonoses. Monograph Series, No. 19, 276 pp. Illust. 15/-; $3.00, Sw. fr. 12. French Edition in preparation. Also published as FAO Agricultural Studies, No. 25. World Health Organization, Geneva, 1953.

Expert Committee on Environmental Sanitation. Third Report. 25 pp. $0.25. World Health Organization, Geneva; The Ryerson Press, Toronto, 1954.

Expert Committee on Rabies. Second Report. 27 pp. $0.25. World Health Organization, Geneva; The Ryerson Press, Toronto, 1954.

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1. McHardy and Brown: Sou. M.J. 45:1139, 1952.
2. Lorber and Shay: Fred. Proc. 12:90, 1953.

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From the Journal of September 1924

"The supplement to the *British Medical Journal* of July 26 contains a full report of the proceedings at the 92nd annual meeting of the British Medical Association. Of particular interest to Canadian readers are the details concerning the visit of Sir Jenner Verrall and Dr. Cox, as delegates to the Canadian Medical Association. The proposals they brought forward, with a view to the affiliation of the Medical Associations of both countries, are by now familiar to the Canadian profession, but there is an additional interest in noting the cordial reception accorded the delegates when they gave an account of their mission.

What did these proposals amount to, it was asked? It was acknowledged, and gladly too, that they were based largely on sentiment; but there were some notable advantages to be secured in the co-operation of the two Associations. A definite advantage to the Canadian profession it is hoped will be derived from their being able to draw on the experience of the older country in regard to the treatment from the professional point of view, of the many industrial problems which will certainly accompany our industrial development."

Nova Scotia News

"Work is now well advanced in the enlargement of the pathological building of the Victoria General Hospital. As the plans call for complete remodelling of the original building, Dr. Nicholls with his staff and equipment are being temporarily housed in a section of the Dalhousie Health Centre. In a few months they will go back to the old site, but to a greatly enlarged building which will represent the most modern ideas in laboratory planning and equipment. Three large floors will be devoted to the clinical and laboratory work of the Victoria General and associated hospitals and to the teaching of pathology and bacteriology, while a fourth floor will be given over to the laboratories of the Department of the Public Health of Nova Scotia. The completion of this building will round out an extensive programme of enlargement and improvement which, within the past few years, has brought the Victoria General into a foremost place in the hospital world."

Manitoba News

"Winnipeg has been favoured this year in the number and quality of her medical visitors. The meetings of the Manitoba Medical Association on June 23 and 24 and the annual dinner of the Association had among the speakers: Sir Jenner Verrall, senior member of the Council of the British Medical Association and Dr. Alfred Cox, secretary, Sir John Thomson-Walker, Prof. W. W. Chipman, Prof. Austin, Dr. F. N. G. Starr, Dr. Geo. Young, Prof. Horst Oertel and Prof. Tait and their memory is still cherished. On July 29 the Winnipeg Medical Society had as their guest of honour at a luncheon, Dr. Elliott P. Joslin of Boston, the noted authority on diabetes.

"Dr. Joslin stated that not to use insulin where it was indicated was as great a crime of omission as failure to use antitoxin in diphtheria. In his own clinic several hours were devoted to the education of his patients. They were instructed as to diets, the significance of sugar in the urine, insulin shock and its remedy. He credited Prof. McLeod of Toronto in having trained an undergraduate, Mr. Best, to that high degree of efficiency where he could participate in a great discovery. A vote of thanks to Dr. Joslin was moved by Dr. E. W. Montgomery and seconded by Dr. C. R. Gilmour.

"Dr. Thos. McCrae of Philadelphia whom we are proud to claim as a Canadian spent two or three hours on July 31, making ward rounds in the Winnipeg General Hospital. A large number of doctors followed the rounds and were pleased with the keenness of his clinical insight."

A Reserve Fund

"Our General Secretary has recently received a communication from one of the Association's members which we here quote in full.

Montreal, July 7, 1924.

'Dear Dr. Routley,

I have long felt the desirability of the Association starting some reverse fund to be maintained and gradually increased, as the years roll, by donations and legacies, the interest of which will be applied in assisting the Association to carry out activities that may tend to the advancement of the profession generally throughout our various provinces, and to the furtherance of all the aims of our Association. A beginning sometime should be made Will you permit me to hand over to you the two bonds I hold against the Association for one hundred dollars each, and to express the hope that I may in the near future be able to add to this very small bequest.

Yours very sincerely.'

"We regret that he dictates the condition that here and now he shall be anonymous.

"His initiative cannot but strike a responsive chord with many. The Association activities have latterly become more and more numerous and more and more varied, and it is essential that special funds be established and reserve funds created. One special fund recently created is the Lister Memorial Fund. This second reserve fund has now its nucleus in the two $100 bonds just transferred to the Association.

"No doubt there are those who when subscribing to the Association's Bond issue considered the money as contributed and not invested. To any such the way has now been pointed which will permanently benefit the Association to the extent of the original intention."

From Annual Meetings Proceedings

"A resolution was also passed at the last meeting of the Executive Committee increasing the Editorial Board allowance by another thousand dollars. This enables the Editor to pay for special work, and to remunerate and thus encourage several younger members of the profession who are devoting considerable time to *Journal* work. The work done by Dr. Malloch and Dr. MacDermot is particularly praiseworthy. The exact distribution of this amount will be placed before the Committee by the Managing Editor."

NEWS AND NOTES

(Continued from page 310)

to devote themselves to research in the basic medical sciences. The awards are open to citizens of the United States and Canada.

Fellowships in Radiological Research are administered for the James Picker Foundation by the Division's Committee on Radiology. The Foundation has expressed particular interest in the support of candidates who propose to carry on research oriented toward the diagnostic aspects of radiology. Appointments are not limited to citizens of the United States.

Applications for 1955-1956 under any of these programmes must be postmarked on or before December 10, 1954. Fellowships are awarded in the early spring. Complete details and application blanks may be obtained from the Fellowship Office, National Academy of Sciences–National Research Council, 2101 Constitution Avenue, N.W., Washington 25, D.C.

EMPLOYMENT OF THE AGING

One effect of medical care has been an increasing aging of the population. The care of the aged sick has become of widespread interest throughout the world, and of increasing difficulty while the healthy aging person is finding it increasingly difficult to obtain employment. One of the difficulties in finding work for the aged is the physical fitness of such persons to perform manual work: as Leggo* points out, much has been said about the loyalty, mature judgment and faithful attendance of the aged but the irreversible changes of age dictate a lessening of physical activity. In those industries where the majority of jobs require moderate exertion and in which the lighter jobs are already filled by those with the necessary experience and qualifications, placement of the aged may be difficult or impossible. The aged labourer cannot be expected to take on semi-skilled employment, but management may find it possible to re-engineer jobs by "effort saving" devices or installations to reduce the physical demands of the job. In the rehabilitation of the aged patient socio-economic status, educational background, skills developed and exercise tolerance are therefore all important: the individual who has been at a desk all his life cannot be expected to show the same tolerance for exercise as his labouring brother but he will probably grasp new ideas much quicker.–W.F.T.T.

STATE DEPARTMENT OF MENTAL HYGIENE EXPLAINS WHY IT USES COMICS, PUPPETS, EVEN A HAUNTED HOUSE TO TEACH MENTAL HEALTH

The philosophy behind a new effective approach to public education is described in a small booklet published by the New York State Department of Mental Hygiene.

In releasing the booklet, Commissioner Newton Bigelow, M.D., indicated that its purpose is to explain "why this department uses comic books, puppet shows, and similar media to teach the principles of good mental health." The report summarizes five years of activity in a new educational programme which attempts to reach the man on the street by speaking his language and using ideas he can grasp and accept.

Discussing the problem of mass education, the author points out that "there is no homogeneity of background, no community of interest, and no established motivation. We must reach out to an assorted body of indifferent human beings, command their attention, enlist their interest, beat down their resistance, and communicate with them in terms that they will understand."

The title, *The Ear of the Beholder*, is taken from a quotation which appears on the title page: "While beauty may be said to lie in the eye of the beholder, it is a scientific truth that sound does not exist save in the ear of the beholder. In much the same way, no words of wisdom, however cogently expressed and vehemently spoken, can ever find their mark unless they are heard and understood."

One of the most successful of the unorthodox devices used in the programme is the Haunted House exhibit first shown at the State Fair last fall. Humorously exploiting the theme "Are You Living in a Haunted House?" the project deals with fear–the basis of most emotional difficulties–pointing out that many of the fears that haunt us are foolish, unfounded, or unnecessary. "The 35-foot exhibit was so constructed," the booklet explains, "that visitors could actually enter a creaking door and prowl through dark passages inhabited by disappearing ghosts, skeletons, bats, spiders, and assorted phantoms. This spine-tingling safari was punctuated by whispering voices which asked the visitors some soul-searching questions. While the thrills and horrors of the haunted house were presented with tongue in cheek, the messages dealt with serious problems. "Are you haunted by ghosts of the past? Is there a skeleton in your closet? Are you afraid of tomorrow?"

The public responded to this unusual educational effort by storming the creaking door. All during the fair long lines of visitors waited patiently for a chance to enter, and many had to be turned away each evening when it was time to close. "We had no choice," Dr. Bigelow said, "but to schedule a repeat appearance of the Haunted House at this year's State Fair in September."

The booklet, which is available to agencies and organizations engaged in mental health education, was written by Margaret M. Farrar, director of the programme.

*LEGGO, C.: *Indust. Med. & Surg.*, **23**: 73, 1954.

PROGNOSIS IN MYOCARDIAL INFARCTION

As in the case of multiple sclerosis, careful studies of the natural history of myocardial infarction continue to show that former pessimism was ill-justified. Cole and his co-workers in Chicago* have recently added another link to the chain of evidence that an attack of myocardial infarction may not be incompatible with a fair span of life.

They studied a carefully selected group of 461 cases of definite first acute myocardial infarction, of which they were able to follow up 390. Of the latter, 105 died within two months of the first attack, this being taken as the period of immediate mortality. Of the remaining 285 two-thirds survived for more than five years, two-fifths lived for over 10 years, and one-tenth were still living after 15 years. Since some are still alive, the final life expectancy in the group will obviously be even better than appears.

The authors have made an analysis of factors affecting prognosis after a first bout of myocardial infarction, and show that males have a slightly better expectation of life than females, probably in part because the former are on an average younger at the first attack. Patients who had hypertension or diabetes mellitus before the attack are less likely to survive for a long period, though they are not more likely to die in the first two months. Previous angina pectoris has little effect on life span. Congestive heart failure, gallop rhythm and pulmonary embolism are of ill omen, but acute diffuse pericarditis has little influence on prognosis. It is interesting to find that tobacco and alcohol have no significant effect on life expectancy, nor has body build. A high nonprotein nitrogen level and a high white cell count are of bad prognosis. It is impossible to forecast survival electrocardiographically or by measurement of heart size. The best guide to the long-term outlook is the degree to which the patient is able to resume activity. A person able to resume full activity has almost a three to one chance of living for over 10 years, but one who remains bedridden has less than a one in ten chance to do so. This finding is not of course to be taken as an indication for forcing patients into an early grave by making them do more than they are able.

Taken all round, the figures presented are heartening, especially when it is remembered that we are dealing mostly with patients already in the 50 to 60 age group, and that a 15-year survival will bring many up to the Biblical allotted span.

MORE PÆDIATRICIANS FOR QUEBEC UNIVERSITIES

The second annual Mead Johnson Awards for post-graduate study in Pædiatrics has just been announced by Messrs Mead Johnson & Company of Canada Limited. A total of $10,000 will be granted to the three Quebec universities and the Fellowship winners this year are as follows: From McGill University—Dr. Albert G. Hewitson, Dr. Arthur J. Markus, Dr. Donald A. Hillman; From University of Montreal—Dr. Jacques April, Dr. Jean-Luc LeBlanc, Dr. Guy Saint-Laurent; From Laval University—Dr. David Jacob, Dr. Yves Cossette, Dr. Pierre Paul Demers, Dr. Fernand Hould.

The Mead Johnson Award Winners have completed their general internship and are now ready to begin a residency in Pædiatrics. In each case the Faculty of Medicine and the Department of Pædiatrics at the universities will act as trustees and administer the funds.

The higher cost of teaching and the need for more specialists puts a severe burden on university budgets. It is this kind of close co-operation between universities and private enterprise that provides the means to keep up with medical progress and train new men to take advantage of it.

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*Cole, D. R., Singian, E. B. and Katz, L. N.: *Circulation*, 9: 321, 1954.

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NEWS AND NOTES

(Continued from page 72 of the advertising section)

COLLEGE OF PHYSICIANS OF PHILADELPHIA

On July 14, 1954, the College of Physicians of Philadelphia awarded the Alvarenga Prize for 1954 to DeWitt Stetten, Jr., M.D., Associate Director in Charge of Research, National Institute of Arthritis and Metabolic Diseases, National Institute of Health, Bethesda, Maryland, for his outstanding contributions to our knowledge fo metabolic diseases.

The Alvarenga Prize was established by the will of Pedro Francisco DaCosta Alvarenga of Lisbon, Portugal, an Associate Fellow of the College of Physicians of Philadelphia, to be awarded annually by the College of Physicians on the anniversary of the death of the testator, July 14, 1883.

COSMETIC ADVERTISING

Advertising of medical products in lay journals often lays stress on the physiological factors available in a specific preparation. Millions of dollars are spent on advertising cosmetics, and the manufacturers of creams containing various hormones have made claims about these products. Behrman in a recent article, has drawn certain conclusions from the clinical study of a group of 31 women. He found no appreciable difference in the effects of an œstrogenic hormone cream used on one side of the face compared with the opposite side which was treated with either the identical base without hormones or a commercial night cream.—Behrman, H. T., *J. A. M. A.*, 135: 119, 1954.

ALCOHOLISM IN MONTREAL

A long delayed report on Alcoholism in Montreal has now appeared in the *Health Bulletin of Montreal* (40: 3, 1954). This report has been produced by a committee on alcoholism appointed by the Montreal Council of Social Agencies who have been deeply concerned with the problem of alcoholism in Montreal and the apparent lack of facilities for its treatment and prevention.

There was difficulty in obtaining an accurate picture of the extent of the problem of alcoholism, due largely to the nature of the disease itself. Figures collected in other centres however suggest that about 3% of those who drink become alcoholics. The Committee undertook a sampling of the community to determine the size of the problem and the extent of its need. Questionnaires of business and industrial firms representing about 30,000 employees showed wide variation as to estimated numbers of problem drinkers. The known and estimated incidence among the male population was 0.7%.

Inquiries of hospitals showed that none of those questioned thought that present facilities were adequate and most of them said they were inadequate. Amongst suggestions for treatment of alcoholics in the city were: provision of beds in general hospitals, or of special wards; beds in mental hospitals; special clinics; use of existing psychiatric clinics; information centres for advice; farms for rehabilitation.

It was felt that alcoholism is a major health and social problem in Montreal even though all the facts are not known. Probably only a small percentage of alcoholics are derelicts; the majority are to be found in the industries, professions, and homes—at all levels of society.

In dealing with alcoholism the co-ordination of existing community resources and the further development of adequate facilities for the treatment and rehabilitation of alcoholics, is of primary importance. The handling of the indigent alcoholic at present comes largely under the jurisdiction of the courts, which is a most undesirable method. It provides partial treatment and protection but does not deal with rehabilitation.

After due consideration of the available information it was concluded that certain minimal community facilities are needed in Montreal for the prevention and treatment of alcoholism. The Committee recommends the formation of an Information and Co-ordination Centre with a minimal staff of a director (preferably medical), a medical social worker and a community research analyst; to supply information about existing facilities; to co-ordinate the work of community agencies interested in alcoholism; and to give leadership in an educational and research programme.

There are clinics for alcoholism at two of the city hospitals now, and it is suggested that these be further developed and others formed. Later, a small number of hospital beds should be made available for in-patient treatment.

The success of any community programme depends on the integration of all available facilities towards the maximum rehabilitation of alcoholics, which should be regarded as a continuous process from the beginning of treatment to final recovery and self-support of the subject.

On the preventive side there should be an educational programme on the prevention control and treatment of alcoholism through all possible media, along with a continuing research programme under university guidance; and finally provision for training of adequate personnel to meet the needs of the programme in the community.

TUMOUR TREATMENT WITH ACTINOMYCIN C

It has been known since 1950 that actinomycin, an antibiotic produced by actinomycetes, has an inhibiting action on growth of certain experimental tumours.[1] In 1952-3 Hackmann[2] reported from Germany an effect of actinomycin C on mouse tumours and a cytostatic effect on the lymphatic system. In 1953, Schulte and Lings,[3] also working in Germany, reported favourable effects of the antibiotic in Hodgkin's disease. Because these results had attracted the attention of the press in France and led to a popular demand for the antibiotic, the French Ministry of Health arranged for a series of clinical trials in Paris, Lyon, Lille, Marseille and other cities in France. Small series of patients were treated by intravenous injections of the order of 300-400 micrograms actinomycin C daily. Toxic effects were, as previously reported in Germany, rarely serious. Patients selected were suffering from such conditions as Hodgkin's disease, lymphosarcoma and bronchial carcinoma. The results of the trials[4] were unfortunately almost uniformly disappointing and in general vastly inferior to those obtained with nitrogen mustards. It should, however, be pointed out that the reports are only preliminary ones. Actinomycin C may yet prove to be a useful adjunct to commoner therapeutic methods.

1. Stock, C. C.: *Am. J. Med.*, 8: 658, 1950.
2. Hackmann, C.: *Strahlenther.*, 90: 296, 1953.
3. Schulte, G. and Lings, H.: *Strahlenther.*, 90: 301, 1953.
4. *Idem*: *Presse méd.*, 62: 737, 1954.

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NEWS AND NOTES

(Continued from page 74 of the advertising section)

WELFARE OF LABORATORY ANIMALS

Some laboratory workers in this country may not be familiar with the activities of the Universities Federation for Animal Welfare, 7a Lamb's Conduit Passage, London, W.C.1, which "seeks the aid of biological research workers and others in fostering in laboratories in Britain and abroad consideration for the physical and mental comfort of experimental animals, avoidance of procedures which involve serious suffering, and the development of techniques calculated to reduce discomfort to a minimum." Their best-known publication is "The UFAW Handbook on the Care and Management of Laboratory Animals," published in 1947. The Federation has now drafted, at the request of foreign scientists, a list of suggestions for the legal protection of laboratory animals, based mainly on British practice. The draft is available from UFAW in pamphlet form, and should prove of interest to biological scientists.

RESISTANCE TO INSECTICIDES

Our most dangerous rivals on this earth, the insects, have in the last decade suffered a serious setback with the development of effective residual insecticides such as DDT, BHC and the organic phosphorus group. From this setback they are now rallying, as shown by the reports from various parts of the world of development of resistance to insecticides in house flies, anophelines, fleas, lice, bedbugs and ticks. Appreciating the threat to public health of this development, the World Health Organization arranged a symposium, jointly with the Istitute Superiore di Sanita, Rome, on the control of insect vectors of disease. This symposium, at which Canada was represented by Prof. A. W. A. Brown of the University of Western Ontario, is now reported in the *Chronicle* of WHO (8: 129, 1954).

The most striking development of resistance has been in the house fly; failure to control this insect is interfering seriously with the world-wide campaign against infant diarrhœa and dysentery. Apart from this, however, resistance among the more important disease vectors has so far been limited, so that the problem is not yet as serious as some pessimists have suggested. Moreover, some of the newer insecticides, including the important organic phosphorus group, promise success in the control of insects resistant to chlorinated hydrocarbons such as DDT and BHC.

The likelihood that insects may acquire resistance to any of the new compounds points the need for increased emphasis on basic principles of sanitation. It is easier to spray a marshy area than to drain it, but as a long-term proposition drainage or filling, flushing of streams and destruction of aquatic vegetation will pay dividends and wherever possible should be an integral part of all insect-control schemes.

The question of biological methods of control also requires investigation. Discovery, culture and dissemination of viruses, bacteria, protozoa or fungi lethal to insects is a distinct possibility needing further study. In view of the large numbers of personnel handling insecticides, there is also an obvious need for study of the toxic effects of the latter on human subjects. Fatalities due to carelessness have already occurred, but have fortunately been rare.

The participants in the symposium conclude their report with suggestions for basic physiological research on insect resistance, including the genetic aspects, and for international co-ordination of studies. The World Health Organization has already done invaluable work in this respect; it must obviously continue to act as an international watchdog over this potential danger to health and wellbeing.

MORE FEDERAL HEALTH AID FOR PROVINCIAL PROJECTS

Approval of further federal health grants to the provinces for a variety of projects, and amounting to more than $150,000 has been announced. The largest current contribution, $75,000 under the Hospital Construction Grant in the National Health Programme, will assist Saskatchewan in increasing accommodation at the government hospital at Weyburn by construction of a new 150-bed nurses' residence.

A Cancer Control Grant of $50,727.26 has been approved to support operation of a cancer diagnostic and treatment centre at St. Joseph's Hospital, Three Rivers, Que. This centre, strategically located between Montreal and Quebec, will fit in with Quebec's province-wide cancer planning and also would be used in postgraduate training for physicians of the area. The federal funds, to be matched by a provincial grant, will assist in staffing and equipping the centre.

It is also announced that Ontario will receive $10,333.33 under the Hospital Construction Grant for an addition which would increase the accommodation by seven active treatment beds and 12 bassinets at the Oakville-Trafalgar Memorial Hospital at Oakville, Ont.

A Child and Maternal Health Grant of $3,800 to Ontario will assist research in Toronto hospitals relating to complications in pregnancy among women of certain blood types. This study will be directed by Dr. D. E. Cannell, head of the Department of Obstetrics and Gynæcology at University of Toronto.

Also announced was a General Public Health Grant of $8,300 to Alberta to support a demonstration programme of nurse recruitment among high school graduates.

(Continued on page 78 of the advertising section)

Dramamine's® Effect in Vertigo

Dramamine has become accepted in the control of a variety of clinical conditions characterized by vertigo and is recognized as a standard for the management of motion sickness. Site of action is probably in the labyrinthine structure.

Vertigo, according to Swartout, is primarily due* to a disturbance of those organs of the body that are responsible for body balance. When the posture of the head is changed, the gelatinous substance in the semicircular canals begins to flow. This flow initiates neural impulses which are transmitted to the vestibular nuclei. From this point impulses are sent to different parts of the body to cause the symptom complex of vertigo.

Some impulses reach the eye muscles and cause nystagmus; some reach the cerebellum and skeletal muscles and righting of the head results; others activate the emetic center to result in nausea, while still others reach the cerebrum making the person aware of his disturbed equilibrium. *Vertigo may be caused by a disease or abnormal stimuli of any of these tissues involved in the transmission of the vertigo impulse, including the cerebellum and the end organs.*

A possible explanation of Dramamine's action is that it depresses the overstimulated labyrinthine structure of the inner ear. Depression, therefore, takes place at the point at which these impulses, causing vertigo, nausea and similar disturbances, originate. Some investigators have suggested that Dramamine may have an additional sedative effect on the central nervous system.

Repeated clinical studies have established Dramamine as valuable in the control of the symptoms of Ménière's syndrome, the nausea and vomiting of pregnancy, radiation sickness, hypertension vertigo, the vertigo of fenestration procedures, labyrinthitis and vestibular dysfunction associated with antibiotic therapy, as well as in motion sickness.

Any of these conditions in which Dramamine is effective may be classed as "disease or abnormal stimuli"* of the tissues including the end organs (gastrointestinal tract, eyes) and their nerve pathways to the labyrinth.

Dramamine (brand of dimenhydrinate) is supplied in tablets of 50 mg. and liquid (12.5 mg. in each 4 cc.). It is accepted by the Council on Pharmacy and Chemistry of the American Medical Association. G. D. Searle & Co., Research in the Service of Medicine.

*Swartout, R., III, and Gunther, K.: "Dizziness:" Vertigo and Syncope, GP *8*:35 (Nov.) 1953.

NEWS AND NOTES

(Continued from page 76 of the advertising section)

FEDERAL GRANTS AID TRAINING OF PATHOLOGISTS FOR NEW BRUNSWICK

Three young physicians are being trained with the aid of federal health grants for service as pathologists in New Brunswick. Approval has been given to Laboratory and Radiological Services grants totalling $7,620 to enable the province to train these doctors, under terms of the National Health Programme, with the aim of improving pathological services in New Brunswick.

Dr. L. Bernier, Quebec City, is beginning a two-year course in pathology at Laval University, training at Hôtel-Dieu and Enfant Jésus Hospitals. He will take another year's training before joining the New Brunswick Provincial Department of Pathology. Dr. C. J. Alexander, Fredericton, N.B., is beginning a three-year course in pathology at the Peter Bent Brigham Hospital, Boston, Mass., before accepting a similar post, and Dr. Alfred B. Bastarache, Shediac, N.B., will study in the provincial laboratories at Saint John for one year before joining the staff under direction of the provincial pathologist.

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WATERBORNE FLUORIDES AND MORTALITY

The United States Public Health Service describes a comparative study of mortality from certain causes in populations drinking almost fluoride-free water (less than 0.25 p.p.m.) or water containing at least 0.70 p.p.m. of fluoride. The authors paired off 32 "non-fluoride" and 32 nearby "fluoride" cities across the United States and studied mortalities from all causes and from five selected causes—heart disease, cancer, intracranial lesions, nephritis and cirrhosis of the liver in these areas. No statistically significant differences were found. This confirms earlier studies limited to smaller areas, in which no detrimental effect of a caries-preventing level of fluoride in the water supply had been demonstrated.—Hagan, T. L. *et al., Pub. Health Rep.*, 69: 450, 1954.

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ALDOSTERONE

Montreal physicians and biochemists were glad to have a visit on August 6 from Dr. Albert Wettstein of Basel, Switzerland, whose many years of work on steroids culminated recently in the isolation in crystalline form of aldosterone, an outstanding event in endocrinology. By the courtesy of the Ciba Company, a lecture on the new hormone by Dr. Wettstein was arranged at the Hôtel-Dieu, Montreal, and after the speaker had been introduced by Dr. Jacques Genest an interested and appreciative audience heard a description of the isolation of aldosterone and the elucidation of its structural formula.

Originally named "electrocortin" by English workers who have collaborated with a Basel team in studies of the new hormone, the latter is now called aldosterone because its molecule contains an aldehyde group. Aldosterone is primarily a mineralocorticoid, with a profound effect on sodium, chloride and water metabolism, but it is now clear that the strict separation of adrenal steroids into mineralocorticoids and glucocorticoids is no longer possible, since all adrenal hormones have something of both in their actions. Thus Simpson estimates the mineralocorticoid effect of aldosterone to be 30 times that of hydrocortisone, whereas the latter has 100 times the glucocorticoid effect of the former.

Dr. Wettstein's observations on synthesis of corticoids by tissue enzymes and by enzymes from micro-organisms gave a glimpse of a fascinating field of study. As usual, the advent of a new hormone has raised more problems than it has solved.

As regards clinical application, it is fair to say that when aldosterone goes into economically possible large-scale production, it will become the hormone of choice in the treatment of Addison's disease.

NEW JOURNAL ON CLINICAL CHEMISTRY

A new periodical, *Clinical Chemistry*, official journal of the American Association of Clinical Chemists, will begin publication in January, 1955. Designed to serve the clinical laboratory worker, the new journal will be devoted to publication of original articles on all aspects of clinical chemistry. It will also supply the only central abstract service covering the field.

Papers submitted for publication should be addressed to Harold D. Appleton, Editor, Clinical Chemistry, Box 123, Lenox Hill Station, New York 21.

Clinical Chemistry will be published bimonthly by Paul B. Hoeber, Inc., 49 E. 33rd Street, New York 16, N.Y. The subscription price will be $8.00 per year in the U.S.A. and countries of the Pan-American Union, $8.50 in Canada and $9.00 elsewhere in the world.

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RESEARCH IN GERONTOLOGY

The Ciba Foundation announces that it will make five awards, each of average value £300, for research relevant to basic problems of ageing, in 1955. Details may be obtained from Dr. G. E. W. Wolstenholme, Director, Ciba Foundation, 41 Portland Place, London W.1., England.

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U.S. FEDERAL RESEARCH GRANTS

The Surgeon General of the Public Health Service, U.S. Department of Health, Education, and Welfare, has announced approval of Federal grants for 1,442 medical research projects, totalling $14,685,671. Four hundred and fifty-nine of the awards, totalling $4,568,073, were for new research projects; 983, totalling $10,117,598, were for continuation of existing projects. The awards were made to scientists in 335 research institutions in the United States and are administered by the National Institutes of Health, research bureau of the Public Health Service. Institutions receiving grants include the National Institute of Arthritis and Metabolic Diseases, National Institute of Neurological Diseases and Blindness, National Cancer Institute, National Institute of Dental Research, National Microbiological Institute, National Heart Institute, and National Institute of Mental Health. The grants award programme covers support of research in the medical and biological sciences, particularly research in the causes and treatment of heart disease, cancer, mental illness, arthritis and other metabolic diseases (such as rheumatoid arthritis, gout, liver diseases, diabetes), the neurosensory and neuromuscular diseases (glaucoma, retrolental fibroplasia, multiple sclerosis, cerebral palsy, epilepsy), diseases of the teeth and oral cavity, and certain infections such as influenza, infectious hepatitis, the common cold, and malaria.

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OCCUPATIONAL CHEST DISEASES

A conference on silicosis and occupational chest diseases jointly sponsored by the McIntyre Research Foundation of Toronto, Canada, and the Saranac Laboratory of Saranac Lake, New York, has been arranged for Monday, Tuesday, and Wednesday, February 7, 8, and 9, 1955, in the Town Hall at Saranac Lake. Doctors, scientists, and businessmen concerned with the problems of occupational chest diseases in all parts of the United States, Canada, and foreign countries are invited to attend.

Anthony J. Lanza, M.D., formerly director of the Institute of Industrial Medicine and now emeritus professor of Industrial Medicine at New York University-Bellevue Medical Center, has been named chairman of the conference. The business arrangements will be handled by Norman R. Sturgis, Jr., to whom communications concerning the conference should be addressed at Saranac Laboratory, Saranac Lake, New York.